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ÖSTERREICHISCHE GESELLSCHAFT FÜR NEUROLOGIE

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Abstractband

Congress Innsbruck
12.–14. März 2025

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22. Jahrestagung

der Österreichischen Gesellschaft für Neurologie

ÖSTERREICHISCHE GESELLSCHAFT FÜR NEUROLOGIE

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23. Jahrestagung

der Österreichischen Gesellschaft für Neurologie

Congress Center Villach

18.–20. März 2026

SAVE THE DATE

Editorial

Liebe Kolleg*innen,

wir dürfen Sie auf die 22. Jahrestagung der Österreichischen Gesellschaft für Neurologie in Innsbruck im März 2025 aufmerksam machen. Im Namen des lokalen Organisationskomitees und des Vorstandes der ÖGN wäre es uns eine Freude, Sie hier vor Ort begrüßen zu dürfen.

Unter dem Motto „Herausforderungen annehmen" ist es uns gelungen, ein umfassendes Programm zu den Themen Schlaganfall, Demenz, Bewegungsstörungen, Epilepsie und Autoimmunerkrankungen zu erstellen und konnten dazu auch ausgewiesene Referenten gewinnen. Der Kongress wird in seinen 6 Plenar- und 14 Parallelsitzungen einen Einblick in die moderne Neurologie des 21. Jahrhunderts geben und besonderes Augenmerk auf aktuelle Entwicklungen, diagnostische Möglichkeiten und innovative Therapieansätze legen und sich auch der Herausforderung der künstlichen Intelligenz stellen.

Die Praxisseminare und Fortbildungskurse zu wichtigen neurologischen Themen ergänzen den Kongress in bewährter Weise und stehen den Teilnehmer*innen auch nach dem Kongress online zur Verfügung.

Die Tagung richtet sich an Neurolog*innen in Ausbildung, an Fachärzt*innen aus dem angestellten und niedergelassenen Bereich, aber auch an Studierende und Kolleg*innen aus anderen Fachgebieten.

Wie auch in den Jahren davor gilt ein besonderes Interesse auch den jungen Neurolog*innen, für die der Kongress eine Plattform zur Präsentation ihrer klinischen und wissenschaftlichen Arbeiten bieten soll. Mit fast 150 Abstract-Einreichungen für Poster, Fallpräsentationen und Kurzvorträgen zeigten die jungen Kolleg*innen bereits im Vorfeld ihr hohes Engagement. Um diesen Einsatz die gebührende Anerkennung zu zollen, erfolgt die Preisverleihung für die besten Beiträge beim „Abend der Gesellschaft" am Donnerstag. Alle eingereichten Abstracts können in diesem Sonderheft nachgelesen werden.

Der Kongress erfreut sich auch der Unterstützung der Industriepartner mit Fachsymposien und großzügigen Ausstellungsflächen. Besucher*innen können auch hier neue Entwicklungen und Therapieansätze erfahren und diese mit Kolleg*innen und Expert*innen diskutieren.

Wir freuen uns auf ein persönliches Wiedersehen in Innsbruck und laden dazu ein, neben dem fachlichen wissenschaftlichen Austausch auch das einzigartige alpine Flair der Stadt zu genießen. ■

Prim. Univ.-Prof. Dr. Stefan Kiechl
(Tagungspräsident)

Priv. Doz.in Dr.in Bettina Pfausler
(Tagungspräsidentin)

Inhalt Abstractband

IMPRESSUM **Herausgeber:** Österreichische Gesellschaft für Neurologie, Univ.-Prof. Priv.-Doz. Dr. Christian Enzinger, MBA, Präsident der ÖGN. **Chefredaktion:** Prim.a Priv.-Doz.in Dr.in Julia Ferrari, Prim. Univ.-Prof. Mag. Dr. Eugen Trinka, FRCP. **Medieninhaber und Verlag:** MedMedia Verlag und Mediaservice GmbH, Seidengasse 9/Top 1.1, A-1070 Wien, Tel.: 01/407 31 11-0, E-Mail: office@medmedia.at. **Aboverwaltung:** Ariana Richtmann, Mail: abo@medmedia.at. **Lektorat:** www.online-lektorat.at • Sprachdienstleistungen. **Layout/DTP:** Nina Pfann. **Projektbetreuung:** Angela Flaggl. **Produktion:** Claudia Lumpi. **Print:** Print Alliance HAV Produktions GmbH, 2540 Bad Vöslau. **Bezugsbedingungen:** Die Zeitschrift ist zum Einzelpreis von 9,50 Euro plus MwSt. zu beziehen. **Druckauflage:** 3.000 Stück. **Grundsätze und Ziele von *neurologisch*:** Kontinuierliche medizinische Fortbildung für Neurologen, Psychiater und Allgemeinmediziner. **Allgemeine Hinweise:** Namentlich gekennzeichnete Beiträge geben die persönliche und/oder wissenschaftliche Meinung des jeweiligen Autors wieder und fallen somit in den persönlichen Verantwortungsbereich des Verfassers. Angaben über Dosierungen, Applikationsformen und Indikationen von pharmazeutischen Spezialitäten müssen vom jeweiligen Anwender auf ihre Richtigkeit überprüft werden. Trotz sorgfältiger Prüfung übernehmen Medieninhaber und Herausgeber keinerlei Haftung für drucktechnische und inhaltliche Fehler. Ausgewählte Artikel dieser Ausgabe finden Sie auch unter www.medmedia.at zum Download.

Fotos: Bettina Pfausler-Anja Koppitsch Photography; Harald Hegen-FotoHoferlbk

Ankündigung

Fallpräsentationen

Die Fallpräsentationen werden im Rahmen der 22. Jahrestagung der Österreichischen Gesellschaft für Neurologie am Freitag, den 14.03.25 von 11:00 – 12:30 Uhr stattfinden.

F01: Subacute onset progressive gait disorder with recurrent falls: A case report

Cerejo C[1], Ellmerer P[1], Leys F[1], Holzknecht E[1], Mahlknecht P[1], Krismer F[1], Heim B[1]
[1] Department of Neurology, Medical University of Innsbruck, Innsbruck, Austria

F02: Das Ungewöhnliche des Seltenen – fulminanter Verlauf von Gangstörung und kognitiver Beeinträchtigung

Potinga I[1], Kalev O[2], Hofstätter J[1], Resch R[1], Schürz N[1], Christian A[3], Fellner F[3], Heidbreder A[1], Helbok R[1], Mitterling T[1]
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[3] 3 Zentrales Radiologie Institut, Kepler Universitätsklinikum, Johannes Kepler Universität Linz, Österreich

F03: Ein seltenes Zusammentreffen: Zwei bestätigte Fälle der Creutzfeld-Jakob-Erkrankung innerhalb einer Woche in einer Wiener Klinik

Mondorf Y[1], Leißer I[1], Szekeres D[1], Lutsenko I[1], Yilmabasar M[1], Lackner P[1]
[1] Klinik Floridsdorf, Wien, Österreich

F04: Steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT)

Auernig G[1], Sommer P[1], Schernthaner R[2], Fertl E[1]
[1] Neurology, Klinik Landstraße, Vienna, Austria
[2] Radiology, Klinik Landstraße, Vienna, Austria

F05: Der Impfung entwischt – schwerwiegende Manifestation einer „Kinderkrankheit"

Ausserer Staubmann G[1], Mitterling T[1], Istratoaie B[1], Ianosi B[1], Böhm V[1], Potinga I[2], Kneidinger M[2], Wimmer S[3], Sonnberger M[3], Heidbreder A[1], Helbok R[1], Kulyk C[1]
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[3] Institut für Neuroradiologie, Kepler Universitätsklinikum, Linz, Österreich

F06: Unklare chronische Durchfälle bei einer Patientin mit Multisystematrophie: Eine diagnostische Herausforderung

Jagusch F[1], Leys F[1], Ellmerer P[1], Djamshidian A[1], Fanciulli A[1], Mahlknecht P[1], Heim B[1], Krismer F[1]
[1] Medizinische Universität Innsbruck, Innsbruck, Österreich

F07: Rezidivierende aseptische Meningoenzephalitis nach Infekten – warum nur?

Schwendinger F[1], Hinteregger D[1], Böhler C[2], Willburger M[2], Werner P[1]
[1] Institut für Akutneurologie und Schlaganfall, Akademisches Lehrkrankenhaus LKH Feldkirch, Feldkirch, Österreich,
[2] Abteilung für Neurologie, Akademisches Lehrkrankenhaus LKH Rankweil, Rankweil, Österreich

F08: Ein unangenehmes „Klicken" im Ohr

Wimmer B[1]
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V01: Kappa free light chain index predicts long-term disease activity and disability accrual in multiple sclerosis

Berek K[1], Schmidauer M[1], Bsteh G[2, 3], Auer M[1], Barket R[1], Berger T[2, 3], Di Pauli F[1], Grams A[4], Hassler M[5], Lenhart L[4], Milosavljevic D[5], Zinganell A[1], Walde J[6], Deisenhammer F[1], Hegen H[1]

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Background: Kappa free light chain (κ-FLC) index predicts short-term disease activity in early multiple sclerosis (MS). The prognostic value of this biomarker over the long-term is unknown.
Objective: To investigate whether κ-FLC index determined at disease onset predicts relapse actitvity and disability accrual during long-term follow-up.
Method: Patients with a first demyelinating event of the central nervous system who had cerebrospinal fluid (CSF) and serum sampling at the Deparment of Neurology of the Medical University of Innsbruck were eligible for inclusion. At baseline, demographics, clinical characteristics as well as the number of T2 hyperintense lesions (T2L) and contrast-enhancing lesions (CEL) on MRI were assessed. During follow-up, the occurrence of relapse, Expanded Disability Status Scale (EDSS) scores and disease-modifying treatment (DMT) were registered. κ-FLC were measured by nephelometry and κ-FLC index calculated as (CSF κ-FLC/serum κ-FLC)/albumin quotient.
Result: Sixty-four patients with a median age at onset of 32 years (25th–75th percentile: 27–39) and a female predominance of 75 % were followed over median of 113 (90–129) months. Forty-six (72 %) patients experienced relapse and 30 (47 %) showed disability accrual. Multivariable Cox regression analysis adjusted for age, sex, disease duration, T2L, CEL, and DMT administration revealed that κ-FLC index independently predicts time to relapse (hazard ratio [HR]: 1.04, LL-CI: 1.0002, p = 0.049, per increase of 10) and disability worsening (HR: 1.06, LL-CI: 1.02, p = 0.008, per increase of 10).
Conclusion: κ-FLC index predicts long-term MS disease activity independently of other risk factors.

V02: 7T 3D-MR spectroscopic imaging of glutathione uncovers oxidative stress signatures in multiple sclerosis patients

Rumbak R[1, 2], Niess E[2], Dal-Bianco A[1, 6], Niess F[2], Strasser B[2], Hingerl L[2], Kloss-Brandstätter A[4], Grabner G[5], Berger T[1, 6], Bogner W[2, 3], Rommer P[1, 6]

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Background: Oxidative stress is frequently discussed as a significant factor in the pathogenesis of multiple sclerosis (MS), yet its mechanisms and endpoints in vivo remain poorly understood. Glutathione (GSH), the main metabolite involved in counteracting oxidative stress, is challenging to assess in vivo due to its low concentrations and technical limitations. Most studies have relied on 3T scanners with limited sensitivity, while only a few have used 7T scanners, reporting reduced GSH levels in MS patients compared to healthy controls. These studies predominantly applied single-voxel spectroscopy, focusing on small brain volumes with mixed tissue types, specific brain regions, and inconsistently including lesions.[1]
Objective: The study aims to compare GSH levels in normal-appearing white matter (NAWM) and normal-appearing gray matter (NAGM) of MS patients with white matter (WM) and gray matter (GM) in healthy controls. Additionally, it examines GSH differences within MS patients (NAWM vs. NAGM) and healthy controls (WM vs. GM).
Method: This study included 12 MS patients (4F/8M, age ± SD: 44 ± 11) and 9 healthy controls (4F/5M, age ± SD: 31 ± 5). The MS cohort (8 RRMS, 4 SPMS) showed no significant differences in GSH to total creatine (tCr) ratios and was analyzed as a single group. Lesions were manually segmented on MP2RAGE and FLAIR images using ITK-Snap. To ▶

exclude lesions, segmented lesion masks were dilated and subtracted from automatically generated NAWM and NAGM using FreeSurfer's SynthSeg. For healthy controls, WM and GM were segmented in the same way, but no subtraction was performed. GSH levels were quantified using Echo-less 3D MR Spectroscopy Imaging (MRSI). Spectra were phase-corrected, averaged, and quantified in LC-Model with a predefined metabolite basis set. Results, expressed as ratios to tCr, were analyzed using Student's t-test after confirming normality and variance.

Results: GSH to tCr ratios differed significantly between groups. MS patients in NAWM and NAGM exhibited lower GSH levels than controls in WM ($p = 0.003$; mean difference ± SD = 0.037 ± 0.010) and GM ($p = 0.044$; 0.021 ± 0.010). Additionally, GSH levels were higher in WM and NAWM compared to GM and NAGM in both controls ($p < 0.001$; 0.055 ± 0.010) and MS patients ($p = 0.001$; 0.039 ± 0.010). The approach was confirmed as our findings ($p = 0.003$; 0.214 ± 0.069) align with previously reported differences in N-acetyl aspartate (NAA) to tCr ratios between NAWM in MS patients and WM in healthy controls. The observed difference in NAA levels between NAWM and NAGM in MS patients ($p = 0.003$; 0.146 ± 0.037) demonstrates the method's sensitivity.

Conclusion: Our findings are the first to demonstrate lower GSH levels in the NAWM and NAGM of MS patients compared to WM and GM of healthy controls using MRSI. These results highlight the potential of the oxidative stress marker GSH to advance diagnostic and therapeutic strategies.

Acknowledgment: This research was funded, in whole or in part, by the Austrian Science Fund (FWF):[10.55776/DFH50].

References:
[1] Srinivasan R, Ratiney H, Hammond-Rosenbluth KE, Pelletier D, Nelson SJ. MR spectroscopic imaging of glutathione in the white and gray matter at 7 T with an application to multiple sclerosis. Magn Reson Imaging. 2010 Feb; 28(2):163–70

V03: Reactive pleocytosis after repeated lumbar puncture: Implications for clinical practice

Schmidauer M[1], Föttinger F[2], Berek K[1], Auer M[1], Barket R[1], Di Pauli F[1], Krajnc N[2], Stichaller L[2], Zaic S[2], Zinganell A[1], Deisenhammer F[1], Walde J[3], Bsteh G[2], Hegen H[1]

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Introduction: Lumbar puncture (LP) is a routine clinical procedure and in some cases repeatedly performed for diagnostic or therapeutic reasons. The impact of repeated LP on cerebrospinal fluid (CSF) findings is not clear.

Objective: To investigate whether repeated LP is associated with reactive pleocytosis and disruption of blood-CSF-barrier function and to determine the role of interval between repeated LP.

Method: Patients with non-inflammatory neurological disease (NIND) and at least 2 consecutive lumbar punctures (LP) were included. Longitudinal changes in CSF white blood cell count (WBC), CSF total protein (TP) and CSF/serum albumin quotient (Qalb) were assessed depending on the time interval between the LP.

Results: A total of 73 patients with a median age of 35 years (25th–75th percentile: 25–45) and a female predominance of 75 % had second LP after 6 (3–19) days. Twenty (27 %) patients developed pleocytosis with an increase of WBC count to 8/µl (6–15) with a maximum of 30/µl. Patients with pleocytosis had LP significantly earlier than patients without pleocytosis, 3.5 (3–7) vs. 7 (3–28) days. The majority of patients (90 %) with CSF pleocytosis had the second LP within 10 days. Further repeated LP in a subgroup of patients revealed similar findings. CSF TP and Qalb slightly increased in patients with pleocytosis.

Conclusion: "Reactive" CSF pleocytosis occurs in approximately one third of patients after repeated LP mostly when performed within the first 10 days.

V04: Dynamic relationship between CSF immune cells and tissue damage markers in multiple sclerosis

Zaic S[1, 2], König T[1, 2], Ponleitner M[1, 2], Gföllner F[1, 2], Silvaieh S[1, 2], Krajnc N[1, 2], Parvizi T[1, 2], Macher S[1, 2], Kornek B[1, 2], Rommer P[1, 2], Leutmezer F[1, 2], Bsteh G[1, 2], Stögmann E[1, 2], Berger T[1, 2], Zrzavy T[1, 2]

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Background and Objective: Multiple sclerosis (MS) is characterized by complex immune-mediated processes leading to demyelination and neurodegeneration. While cerebrospinal fluid (CSF) biomarkers can track tissue damage, the relationship between specific immune cell populations and tissue damage markers remains poorly understood. We aimed to characterize CSF immune profiles and their association with neurofilament light chain (NfL) and glial fibrillary acidic protein (GFAP) across different MS clinical phenotypes and disease activity states.
Method: We performed comprehensive immunophenotyping of CSF samples from 63 participants (29 relapsing-remitting MS [RRMS], 7 primary progressive MS [PPMS], 27 controls) using spectral flow cytometry. CSF levels of NfL and GFAP were measured using single molecule array technology. Relationships between immune cell populations and biomarkers were assessed using partial correlation and multiple regression analyses, adjusting for age and sex.
Results: RRMS patients showed significant expansion of lymphocyte populations compared to controls, with increased absolute counts of CD3+ T cells (+5062 cells/mL, $p < 0.001$) and CD19+ B cells (+180 cells/mL, $p < 0.001$). In PPMS, we observed elevated frequencies of CD14+CD16+ non-classical monocytes compared to RRMS (+9.9 %, $p = 0.005$). Classical CD14+CD16- monocytes significantly correlated with GFAP levels in controls ($\rho SP = 0.563$, $p = 0.008$), explaining 28.4 % of GFAP variance in regression analysis ($\beta = 0.333$, SE = 0.094, $p = 0.002$). During active RRMS relapse, naive CD4+ T cells showed the strongest association with NfL, with significantly different relationships during relapse ($\beta = 1.062$, $p < 0.001$) vs. non-relapse ($\beta = -0.372$, $p < 0.001$) states. CD8+ T cells demonstrated an inverse relationship pattern, showing positive association with NfL during non-relapse ($\beta = 0.038$, $p = 0.060$) that reversed during relapse ($\beta = -0.066$, $p = 0.016$). CD19+ B cells showed an enhanced positive association with NfL during relapse ($\beta = 0.440$, $p = 0.028$).
Discussion: This study identifies distinct immunological signatures in MS and demonstrates disease activity dependent associations between specific immune cell populations and markers of tissue damage. The relationship between classical monocytes and GFAP in controls suggests a previously unrecognized role for myeloid cells in physiological CNS homeostasis.

V05: Risdiplam in adults with 5q-associated spinal muscular atrophy: A nationwide retrospective observational study in Austria

Keritam O[1, 2], Erdler M[3], Fasching B[1, 2], Zulehner G[1, 2], Rath J[1, 2], Krenn M[1, 2], Gruber V[1], Langweil N[1], Griedl T[4], Kiss C[4], Wanschitz J[5], Hotter A[5], Kleinveld V[5], Horlings C[5], Troger J[6], Grinzinger S[7], Müller P[8], Langenscheidt D[9], Rappold M[10], Wiesenhofer A[10], Gosk-Tomek M[10], Knipp F[10], Mahal S[10], Bernert G[10], Baumann M[11], Zimprich F[1, 2], Topakian R[8], Eggers C[12], Quasthoff S[4], Löscher W[5], Cetin H[1, 2]

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Introduction: Homozygous deletions or (likely) pathogenic variants of the SMN1 gene lead to loss of function of the survival of motor neuron (SMN) protein, thereby causing spinal muscular atrophy (SMA). The natural course of SMA is marked by the continuous progression of motor function deficits. Risdiplam, a small molecule that enhances SMN protein production via the SMN2 pre-mRNA, was approved for SMA treatment by the European Commission in 2021. However, clinical trial data have primarily focused on pediatric patients, with evidence for the efficacy and safety of risdiplam in adults limited to case series and small single-center studies.
Aim and Method: This nationwide retrospective observational study aimed to assess the efficacy and safety of risdiplam in adult patients (≥ 18 years) with 5q-associated SMA. Only patients who were treatment-naive prior to receiving risdiplam were included. Patients were assessed for the availability of functional scores, including the Hammersmith Functional Motor Scale Expanded ▶

(HFMSE, primary endpoint), the Revised Upper Limb Module, and the Amyotrophic Lateral Sclerosis Functional Rating Scale Revised (RULM and ALSFRSr as secondary endpoints) at treatment initiation (baseline, T0) and at least 1 time point ≥ 3 months thereafter. A clinically meaningful improvement in HFMSE and RULM was defined as an increase of ≥ 3 and ≥ 2 points, respectively. Different time points were defined to investigate changes from baseline: 3 months ≤ T1 < 6 months; 6 months ≤ T2 < 12 months; 12 months ≤ T3 < 18 months; T4 ≥ 18 months. Trajectories of changes from baseline were analyzed by linear regression.

Results: A total of 128 patients treated at all Austrian neuromuscular centers were screened for eligibility, with 71 patients receiving risdiplam as their first disease-modifying therapy. Data for HFMSE, RULM, and ALSFRSr were available at T0 and at least 1 other time point for 40 patients (56.3 %), 52 patients (73.2 %), and 43 patients (60.6 %), respectively. HFMSE scores significantly improved at T1 (mean 1.0, SD 2.5, p = 0.0100), T2 (mean 1.0, SD 2.0, p = 0.0132), T3 (mean 1.8, SD 2.8, p = 0.0008), and T4 (mean 1.7, SD 3.1, p = 0.0049) as compared to baseline. A clinically meaningful improvement in HFMSE scores was observed in 16.7 % of the cohort at T1, in 23.3 % at T2, in 29.6 % at T3, and in 30.8 % at T4. The proportion of patients with a clinically meaningful improvement in RULM scores was higher (33.3 % at T1, 35.0 % at T2, 48.6 % at T3, and 52.9 % at T4). The average rate of HFMSE increase was 0.09 per month (95%-CI 0.06-0.11, p < 0.0001). These results were supported by significant improvements of the secondary endpoints RULM and ALSFRSr. The safety profile corresponded to the profile already established for risdiplam.

Summary: The results of this nationwide study provide the first real-world data in Austria on adult SMA patients treated with risdiplam, demonstrating clinical stabilization or motor function improvement in the majority of patients following treatment. This is particularly important, as the natural history of SMA typically involves continuous deterioration of motor function over time, highlighting the significance of our findings.

Acknowledgement: This study was financially supported by Roche.

V06: A retrospective multicenter study on clinical and serological parameters in patients with MuSK myasthenia gravis with and without general immunosuppression

Koneczny I[1, 2], Damas M[1], Zong S[1], De Haas S[1], Huda S[3, 4], van Kruining D[5], Damoiseaux J[6], De Rosa A[7], Maestri M[7], Guida M[7], Molenaar P[1], Van Damme P[8, 9], Fichtenbaum A[2, 10], Perkmann T[10], De Baets M[1], Lazaridis K[11], Zouvelou V[12], Tzartos S[11, 13], Ricciardi R[7, 14], Losen M[1], Martinez-Martinez P[1]

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Introduction: Muscle-specific kinase (MuSK) myasthenia gravis (MG) is caused by pathogenic autoantibodies against MuSK that correlate with disease severity and are predominantly of the IgG4 subclass. The first-line treatment for MuSK-MG is general immunosuppression with corticosteroids, but the effect of treatment on IgG4 and MuSK IgG4 levels has not been studied.

Method: We analyzed the clinical data and sera from 52 MuSK-MG patients (45F, 7M, median age 49 [range 17–79] years) from Italy, the Netherlands, Greece, and Belgium, and 43 AChR-MG patients (22F, 21M, median age 63 [range 2–82] years) from Italy, receiving different types of immunosuppression, and sera from 46 age- and sex-matched non-disease controls (with no diagnosed diseases, 38F, 8M, median age 51.5 [range 20–68] years) from the Netherlands.

We analyzed the disease severity (assessed by MGFA or QMG score) and measured concentrations of MuSK IgG4, MuSK IgG, total IgG4 and total IgG in the sera by ELISA, RIA, and nephelometry.

Results: We observed that MuSK-MG patients showed a robust clinical improvement and reduction of MuSK IgG after therapy, and that MuSK IgG4 concentrations, but not total IgG4 concentrations, correlated with clinical severity. MuSK IgG and MuSK IgG4 concentrations were reduced after immunosuppression in 4/5 individuals with before-after data, but data from non-linked patient samples showed no difference. Total serum IgG4 levels were within the normal range, with IgG4 levels above threshold (1.35g/L) in 1/52 MuSK-MG, 2/43 AChR-MG patients, and 1/45 non-disease controls. MuSK-MG patients improved within the first 4 years after disease onset, but no further clinical improvement or reduction of MuSK IgG4 were observed 4 years later, and only 14/52 (26.92 %) patients in total, of which 13 (93.3 %) received general immunosuppression, reached clinical remission.

Discussion: We conclude that MuSK-MG patients improve clinically with general immunosuppression but may require further treatment to reach remission. Longitudinal testing of individual patients may be clinically more useful than single measurements of MuSK IgG4. No significant differences in the serum IgG4 concentrations and IgG4/IgG ratio between AChR- and MuSK-MG patients were found during follow-up. Further studies with larger patient and control cohorts are necessary to validate the findings.

V07: Health-related quality of life after spontaneous subarachnoid hemorrhage: A prospective cohort study

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Background: Impaired health-related quality of life (HR-QoL) is common after spontaneous subarachnoid hemorrhage (SAH).

Research question: Here, we aimed to describe the prevalence of HR-QoL impairment 1 year after SAH and to identify associated factors.

Method: In this prospective observational study, HR-QoL was assessed in 183 patients 1 year after SAH. We used the Short-Form-36 (SF-36) questionnaire, which consists of 8 health domains that can be subdivided into mental and physical health components (MCS, PCS). Participants responded to scales on subjective attention deficit, mental health symptoms, and fatigue. Functional outcome was assessed with the modified Rankin Scale (mRS). Using multivariable regression analysis, we identified factors associated with impaired HR-QoL (MCS or PCS < 40).

Results: Patients were 53 years of age (IQR, 46–61) and presented with a median Hunt and Hess score of 2 (2–3). HR-QoL was impaired in 66/183 patients (36 %) with the highest abnormality in physical and emotional roles. A lower Hunt and Hess score ($p = 0.036$), female sex ($p = 0.017$), self-reported depression ($p = 0.001$), fatigue ($p < 0.001$), and reduction of drive ($p = 0.019$) were associated with overall impaired HR-QoL and explained 68.9 % of the observed variance. 26 % (n = 48) scored below the normal range on the MCS, and independent associations emerged for self-reported anxiety and depression, fatigue, and reduction of drive. Impairments in the PCS were reported by 35 (19 %) patients, and independent associations were found for worse 3-month functional outcome and fatigue.

Conclusion: One in three patients reported a reduction in HR-QoL 1 year after SAH, with an important impact on HR-QoL of mental health problems and fatigue.

V08: Feasibility and clinical relevance of using lomustine and temozolomide combined with tumor-treating fields in newly diagnosed grade 4 astrocytoma

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Background: Since the publication of the CeTeG/NOA-09 trial1 in 2019, the combination of lomustine (CCNU) and temozolomide (TMZ) for IDH wildtype glioblastoma with methylated MGMT promoter (mGB) has variably been used as a first-line treatment option. According to EF-14 trial2 and post-marketing surveillance registries, the use of tumour treating fields (TTF) in newly diagnosed glioblastoma has been shown to be feasible, safe, and to significantly increase overall survival (OS) and progression-free survival (PFS). However, combining CCNU and temozolomide with TTF therapy in newly diagnosed grade 4 astrocytoma has not yet been extensively studied.

Research question: Is chemotherapy according to CeTeG protocol combined with TTF therapy in the adjuvant setting a feasible and clinically meaningful treatment option for patients with grade 4 astrocytoma?

Method: A retrospective data analysis of adult patients treated for newly diagnosed glioblastoma with methylated MGMT promotor (2016 CNS WHO classification) using CCNU and TMZ concomitantly during radiation therapy and combined with adjuvant TTF therapy thereafter was used for survival analysis. Furthermore, the correlation between median daily usage and PFS/OS was analyzed. Only patients treated with TTF for at least 8 weeks were included in this analysis.

Results: Eighteen patients with mGB and 2 patients with methylated, IDH mutant astrocytoma grade 4 treated at a tertiary medical center between 6/2018 and 12/2024 were included in this analysis. At the data cutoff (December 20 2024), 10 patients were still on TTF therapy. The median treatment duration of TTF therapy in the entire cohort was 14.5 months, with a median daily usage of 81 %. The median duration of follow-up was 536.5 days. The median PFS in our cohort was 14.6 months, while the median OS was 16.2 months. The overall duration of TTF therapy correlated strongly and significantly with longer PFS and OS. However, no significant correlation between daily usage (in percent) and PFS/OS was found in our analysis. During the temozolomide/lomustine and TTF therapy, 2 haematological adverse events (CTCAE grade 3) were reported.

Conclusion: Combining TTF therapy with a combination of CCNU and temozolomide is feasible, and patients adhere to the treatment plan well with a median daily usage of 81 %. Longer duration of TTF therapy correlated strongly and significantly with longer PFS and OS, underscoring the importance of this additional treatment option. Due to the low sample size of 20 patients with 10 patients still undergoing treatment at the time of cutoff, no significant correlation between daily usage and PFS/OS could be found. However, with this treatment regimen, some patients survived for more than 5 years over the course of our follow-up. To find a reliable correlation between daily usage (in percent) of TTF therapy and PFS/OS using this combination of treatment modalities, further studies with larger sample sizes are needed in the future.

V09: Der Einfluss eines nahtlosen Rehabilitationsanschlusses nach der Stroke Unit auf die Prognose von Schlaganfallpatient*innen

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Einleitung: Die frühe Rehabilitation nach Schlaganfall ist mit einer Verbesserung der Prognose assoziiert. Ein lückenloser Rehabilitationsprozess ausgehend von der Stroke Unit wird von nationalen und internationalen Leitlinien empfohlen, wobei zugrundeliegende Daten limitiert sind und die Umsetzung versorgungsstrukturelle Herausforderungen birgt.

Ziel: Diese Arbeit untersucht den Einfluss eines nahtlosen Rehabilitationsanschlusses nach der Stroke Unit auf die Prognose von Schlaganfallpatient*innen.

Methoden: Die Studie basiert auf pro-

spektiv gesammelten Daten aus dem österreichischen Schlaganfallregister und inkludierte alle Schlaganfallpatient*innen, die an einer steirischen Stroke Unit zwischen 2012 und 2023 behandelt wurden und eine stationäre Neurorehabilitation innerhalb von 3 Monaten nach Schlaganfall erhielten. Ausgeschlossen wurden Patient*innen mit einem prämorbiden Score nach der modifizierten Rankin Skala (mRS) von > 1, vorbestehender Multimorbidität (definiert als > 2 chronische Erkrankungen), einem mRS-Score von < 3 bei Stroke-Unit-Entlassung sowie mit fehlendem 3-Monats-Follow-up. Als nahtloser Rehabilitationsanschluss wurde ein Direkttransfer von der Stroke Unit in eine spezialisierte B- oder C-Phase-Rehabilitationseinheit definiert. Als primärer Outcome wurden eine mRS-Verbesserung von ≥ 1 von Stroke-Unit-Entlassung bis zum Follow-up gewählt und Prädiktoren mit uni- und multivariabler Statistik analysiert.

Ergebnisse: Die finale Studienkohorte bestand aus 2.497 Patient*innen (medianes Alter: 74 Jahre; weiblich: n = 1.117, 44,7 %). Der mediane mRS-Wert bei Entlassung von der Stroke Unit lag bei 4.Eine mRS-Verbesserung bis zum Follow-up konnte bei 1.663 (66,6 %) Patient*innen festgestellt werden. In der multivariablen Analyse waren ein niedrigeres Alter, männliches Geschlecht, fehlendes Vorliegen eines Diabetes mellitus oder eines früheren Schlaganfalles, ein höher NIHSS-Wert bei Entlassung sowie der Direkttransfer in eine Rehabilitationseinheit mit einer mRS-Verbesserung assoziiert (alle p < 0,01).Die adjustierte Odds Ratio für eine mRS-Verbesserung bei direktem Transfer von der Stroke Unit in eine Rehabilitationseinheit lag bei 1,55 (95 %-Konfidenzintervall 1,25–1,90).**Konklusion:** Der nahtlose Rehabilitationsanschluss nach der Stroke Unit ist mit einer höheren Wahrscheinlichkeit für funktionelle Verbesserung innerhalb von 3 Monaten nach Schlaganfall verbunden. Diese Ergebnisse unterstützen die Entwicklung und Umsetzung eines kontinuierlichen Rehabilitationspfades nach Schlaganfall.

V10: Evaluierung von Delir-Screening Tools bei akuten Schlaganfallpatient*innen: Eine vergleichende Analyse der diagnostischen Leistungsfähigkeit

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Hintergrund: Ein routinemäßiges Delir-Screening wird in Konsensus-Leitlinien empfohlen, um die Früherkennung und Behandlung bei Patient*innen mit akutem Schlaganfall zu verbessern. Es liegen jedoch nur wenige Daten über die diagnostische Leistungsfähigkeit etablierter Screening Tools bei Schlaganfallpatient*innen vor.

Fragestellung: In dieser Studie untersuchten wir daher die Effektivität von 5 häufig verwendeten Delir-Screening-Tools bei Patient*innen mit akutem Schlaganfall.

Methode: Über einen Zeitraum von 1 Jahr wurden alle akuten Schlaganfallpatient*innen, die auf der Stroke Unit des Universitätsklinikums Graz aufgenommen wurden, prospektiv erfasst. Ein kontinuierliches Delir-Screening wurde von geschultem Pflegepersonal durchgeführt und alle 12 Stunden mittels 4 A's Test (4AT), Confusion Assessment Method (CAM), Delirium Observation Scale (DOS), Intensive Care Delirium Screening Checklist (ICDSC) und Nursing Delirium Screening Scale (NU-DESC) dokumentiert. Die Qualität und Nichtunterlegenheit dieser Tests wurde gegen den Goldstandard bewertet, der Diagnose basierend auf den Kriterien des Diagnostic and Statistical Manual of Mental Disorders (Fifth Edition – DSM-5). Diese wurde zweimal täglich, verblindet gegenüber den Pflege-Daten, von den behandelnden Ärzt*innen evaluiert.

Ergebnisse: Von 450 akuten Schlaganfallpatient*innen (mittleres Alter: 74 Jahre, IQR: 18; weiblich: 43,1 %) wurde bei 53 (11,8 %) ein Delir entsprechend den DSM-5-Kriterien diagnostiziert. Das Delir trat im Median 25 Stunden (IQR: 34) nach Aufnahme auf. Während alle getesteten Screening-Instrumente eine hohe Spezifität für die Erkennung eines Delirs aufwiesen (90–97 %), erreichte nur der NU-DESC eine hohe Sensitivität von 87 % (alle anderen Tests: ≤ 66 %) und war einer DSM-5-basierten Diagnose nicht unterlegen (p > 0,05).

Zusammenfassung: Während die meisten gängigen Delir-Screening-Tools eine unzureichende Sensitivität aufweisen, stellt der NU-DESC eine vielversprechende Option für das Delir-Screening bei akuten Schlaganfallpatient*innen dar.

V11: Dysphagie als unabhängiger Risikofaktor für Post-Stroke Fatigue

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Hintergrund: Dysphagie nach einem ischämischen Schlaganfall ist weit verbreitet und hat erhebliche Auswirkungen auf die Mortalität und Lebensqualität von Betroffenen. In dieser Studie untersuchen wir, ob Dysphagie auch das Risiko für Post-Stroke Fatigue erhöht.

Fragestellung: Besteht ein Zusammenhang zwischen Dysphagie und Fatigue bei Patient*innen mit akutem ischämischem Schlaganfall?

Methode: Wir analysierten Daten aus dem STROKE-CARD-Registry (2020–2023), in dem 882 Patient*innen mit akutem ischämischem Schlaganfall rekrutiert wurden. Dysphagie wurde durch eine klinische Schluckuntersuchung zum Zeitpunkt der Aufnahme diagnostiziert. Post-Stroke Fatigue wurde mit der Fatigue Severity Scale (FSS) in Nachuntersuchungen innerhalb des ersten Jahres nach dem Schlaganfall erfasst.

Ergebnisse: Von den 882 Patient*innen hatten 22,0 % zu Beginn der stationären Behandlung eine Dysphagie, die in 16,2 % bis zur Entlassung persistierte. Die Prävalenz von Post-Stroke Fatigue betrug 52,2 % und war bei Patient*innen mit Dysphagie signifikant höher (68,4 % vs. 49,0 %, $p < 0,001$). Die Fatigue-Rate stieg proportional mit dem Schweregrad der Dysphagie und war bei Betroffenen mit schwerer Dysphagie am höchsten (86,7 %, $p < 0,001$). Nach Anpassung an andere Einflussfaktoren war Dysphagie weiterhin unabhängig mit einem erhöhten Risiko für Post-Stroke Fatigue assoziiert (Odds Ratio: 2,03; 95%-KI: 1,22–3,38).

Zusammenfassung: Dysphagie ist häufig nach ischämischem Schlaganfall und erhöht das Risiko für Post-Stroke Fatigue. Eine intensivierte Behandlung von Dysphagie könnte helfen, Fatigue zu reduzieren und somit die Lebensqualität der Patient*innen zu verbessern.

V12: Prädiabetes und Diabetes mellitus Typ 2 ein Jahr nach einem akuten ischämischen Schlaganfall

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Hintergrund: Der Verlauf des glykämischen Status ist bei Schlaganfallpatient*innen bisher nur wenig untersucht, insbesondere im Hinblick auf neue Behandlungsmöglichkeiten für Diabetes mellitus Typ 2 (DM2) wie Glucagon-like Peptide-1-Rezeptoragonisten (GLP-1-RA) und Natrium-Glukose-Co-Transporter-2-(SGLT-2-)Inhibitoren, die bislang nicht spezifisch im Kontext des ischämischen Schlaganfalls untersucht wurden.

Fragestellung: Die Studie untersucht, wie sich der glykämische Status von Schlaganfallpatient*innen im Laufe eines Jahres entwickelt, welche Faktoren mit dieser Veränderung zusammenhängen, welche aktuellen Studien mit GLP-1-RA und SGLT-2-Inhibitoren in dieser Patientenpopulation durchgeführt werden und wie viele Patient*innen die Einschlusskriterien für eine rezente Studie mit Semaglutid bei Nichtdiabetiker*innen erfüllen würden.

Methode: 884 aufeinanderfolgende Patient*innen mit ischämischem Schlaganfall, die in das prospektive STROKE-CARD-Register aufgenommen wurden, wurden hinsichtlich ihres Blutzuckerstatus (Normoglykämie, Prädiabetes, DM2) über einen Zeitraum von 1 Jahr untersucht. Eine multivariate logistische Regression wurde durchgeführt, um Faktoren zu identifizieren, die mit einem Übergang von Normoglykämie zu Prädiabetes oder DM2 verbunden sind. Zusätzlich überprüften wir laufende klinische Studien zu GLP-1-RA und SGLT-2-Inhibitoren im Zusammenhang mit dem akuten ischämischen Schlaganfall.

Ergebnisse: Zu Beginn wiesen 44,6 % (n = 394) der Patient*innen Normoglykämie, 33,9 % (n = 300) Prädiabetes und 21,5 % (n = 190) DM2 auf. Nach 1 Jahr nahm die Normoglykämie um 12,1 % ab (n = 107), während Prädiabetes und DM 2 um 10,2 % (n = 90) bzw. 1,9 % (n = 17) zunahmen. Die Statintherapie war

der einzige signifikante Risikofaktor für die Entwicklung von Prädiabetes oder DM2 innerhalb eines Jahres. Insgesamt hätten 23,4 % (n = 207) unserer Kohorte die Einschlusskriterien für eine kürzlich durchgeführte Studie zu Semaglutid bei adipösen Nichtdiabetiker*innen mit kardiovaskulären Vorerkrankungen erfüllt. Allerdings zielt nur 1 laufende Studie darauf ab, das kurz- und mittelfristige kardiovaskuläre Risikomanagement bei Schlaganfallpatient*innen zu evaluieren.

Zusammenfassung: Angesichts der hohen Prävalenz und der fortschreitenden Entwicklung von Prädiabetes und DM2 bei Schlaganfallüberlebenden besteht ein dringender Bedarf an klinischen Studien, die den Einsatz von GLP-1-RA und SGLT-2-Inhibitoren in dieser Patientengruppe untersuchen.

V13: The diagnostic value of additional neurological symptoms in differentiating functional tremor from non-functional tremor

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Background: Functional tremor (FT) is the most common functional movement disorder (FMD). However, FMD including FT is frequently part of a complex syndrome comprising both motor and non-motor symptoms rather than an isolated symptom.

Objective: This study aimed to evaluate whether FT can be differentiated from non-functional tremor (nFT) based on the findings from a basic neurological examination.

Method: We performed a cross-sectional study in 28 patients with FT and 27 patients with nFT (essential tremor, n = 10; tremor associated with dystonia, n = 18). All patients completed demographic questionnaires and underwent a comprehensive neurological examination. Additional symptoms such as paresis, sensory disturbances, or gait abnormalities—symptoms not explicitly reported by the patients—were assessed. Tremor and dystonia, being the primary complaints, were excluded from this analysis. Binary logistic regression was used to examine the relationship between the presence of additional symptoms and the likelihood of being diagnosed with FT.

Results: Among the 55 patients, 8 patients (FT: 7/28, nFT: 1/27) presented with 1 additional symptom and 10 patients (FT: 9/28, nFT: 1/27) exhibited 2 or more additional symptoms. In contrast, 12/28 patients with FT and 25/27 patients with nFT had no additional symptoms. Specific findings in patients with FT included tubular visual field (1/28), paresis (4/28), sensory symptoms (7/28), gait disorders (9/28), and impaired tandem gait (9/28). In patients with nFT, however, these symptoms were rare, with only 1/27 patient presenting with gait disorder and 2/27 with impaired tandem gait. The presence of additional symptoms was strongly associated with the diagnosis of FT (1 additional symptom: ß = 2.7, p = .017; two or more additional symptoms: ß = 2.9, p = .008). The odds of an FT diagnosis increased by 14.6 with the presence of 1 additional symptom and by 18.8 with the presence of 2 or more additional symptoms. The model´s area under the curve (AUC) was 0.75.

Conclusion: In addition to the clinical diagnosis of FT based on positive signs, identification of neurological signs not explicitly reported by patients may help to distinguish FT from nFT.

V14: Charakterisierung von Gangprofilen und Alltagsaktivität von Patient*innen mit atypischen Parkinsonsyndromen

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Hintergrund: Patient*innen mit atypischen Parkinsonsyndromen (APS), wie der Multisystematrophie (MSA) und der progressiven supranukleären Blickparese (PSP), sind gekennzeichnet durch eine rasche Progredienz sowie eine frühe Einschränkung der Mobilität. Dies führt zu häufigen Stürzen und einer Beeinträchtigung der Lebensqualität für Betroffene und deren Angehörige. Da es sich bei den APS um seltene Erkrankungen handelt, ist unser Wissen über Gang- und Mobilitätsprofile begrenzt, insbesondere über die körperliche Aktivität im täglichen Leben.
Fragestellung: Ziel dieser Studie ist es, Gangprofile unter standardisierten (Lab) und nichtstandardisierten (Home) Bedingungen einer großen Kohorte von Patient*innen mit MSA, PSP und der Parkinson-Krankheit (PK) durch klinische Skalen, Patient*innen-orientierte Fragebögen sowie sensor-basierte Ganganalyse umfassend zu charakterisieren.
Methode: Diese Analyse präsentiert Baseline-Daten aus einer multizentrischen, randomisiert kontrollierten Studie zu Physiotherapie bei Patient*innen mit APS. Patient*innen wurden unter stabiler Medikation und ohne die Mobilität beeinträchtigende Begleiterkrankungen eingeschlossen. In 2 klinischen Visiten wurden motorische und kognitive Skalen sowie Fragebögen zu Aktivität und Lebensqualität erhoben. Während der zweiten Visite wurde zusätzlich eine sensorbasierte Ganganalyse (IGA) durchgeführt. Zwischen den beiden Visiten erhielten die Teilnehmer*innen identische Sensoren für 1 Woche mit nach Hause, um die körperliche Aktivität (PAM) in ihrem Alltag zu überwachen. Klinische, IGA- und PAM-Parameter wurden in den 3 Gruppen verglichen und korreliert.
Ergebnisse: Von insgesamt 106 Patient*innen wurden 84 vollständige Datensätze in die Analyse eingeschlossen (23 MSA, 20 PSP und 41 PD). Während APS-Patient*innen eine kürzere Krankheitsdauer aufwiesen, zeigte sich eine schwerere motorische Beeinträchtigung im Vergleich zur PK (MDS-UPDRS III: MSA 39,0 vs. PSP 40,5 vs. PK 18,0; $p < 0{,}001$). APS-Patient*innen wiesen eine geringere Ganggeschwindigkeit während der IGA sowie eine höhere Variabilität und Asymmetrie der meisten Sensorparameter auf. Im täglichen Leben zeigten die APS-Patient*innen neben einer geringeren Schrittzahl pro Tag auch weniger Aktivität, eine geringere Intensität und einfachere Mobilitätsmuster. Sowohl bei den Sensor- als auch bei den klinischen Parametern wurden krankheitsspezifische Merkmale festgestellt, die MSA und PSP voneinander unterscheiden. Außerdem wurden in allen Gruppen starke Korrelationen zwischen den klinischen Scores und den IGA- und PAM-Parametern festgestellt.
Zusammenfassung: Diese umfassende Charakterisierung von Patient*innen mit MSA, PSP und PK zeigt uns nähere Einblicke in Gang- und Mobilitätsprofile, die multidimensional durch klinische Skalen, Fragebögen, standardisierte IGA und unbeaufsichtigte PAM bewertet wurden. Die Ergebnisse bieten der Forschung neue Möglichkeiten, tiefere Einblicke und ein besseres Verständnis dieser Aspekte bei Patient*innen mit APS.

V15: Ressourcen der österreichischen neurologischen und psychiatrischen Krankenhausabteilungen für Amyloid-Antikörper-Therapien der frühen Alzheimer-Krankheit – eine Erhebung der Österreichischen Alzheimer-Gesellschaft

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die Vorstandsmitglieder der ÖAG und die Leiter*innen der österreichischen neurologischen und psychiatrischen Krankenhausabteilungen

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Hintergrund: Neue krankheitsmodifizierende Therapien bei leichter kognitiver Störung und Demenz durch Alzheimer-Pathologie bedeuten für Betroffene sowie für Neurologie und Psychiatrie einen Fortschritt, aber auch inhaltliche und organisatorische Herausforderungen. Die Therapie-Einschlusskriterien beinhalten Anamnese, klinische und neuropsychologische Untersuchung, Routine-Labor, MRT, Amyloid-PET und/oder Liquor-Untersuchung (degenerative Demenzparameter) sowie Apo-E-ε-Varianten-Status. Nur max. 15–20 % von Personen mit leichter kognitiver Störung und Demenz kommen für eine Therapie infrage. Die Behandlung erfolgt in 2- bis 4-wöchigen Intervallen mittels Infusionen, unter regelmäßigen

klinischen MRT-Kontrollen, und ist spitalbasiert.

Fragestellung: Es ist daher eine Bestandsaufnahme der diagnostischen und therapeutischen Ressourcen/Kapazitäten erforderlich.

Methode: Der Vorstand der Österreichischen Alzheimer-Gesellschaft erarbeitete einen online-basierten Fragenkatalog (Programmierung/Betreuung E. B.) für Leiter*innen aller österreichischen neurologischen (N) und psychiatrischen (P) Kliniken/Spitalsabteilungen sowie der geriatrischen Klinik PMU Salzburg und des Psychosozialen Dienstes Wien. Die Befragung wurde 2023 abgeschlossen und von 2 Studierenden der JKU Linz ausgewertet (E. E., J. M.). Die Ergebnisse wurden nach Plausibilitätsprüfung deskriptiv statistisch ausgewertet.

Ergebnisse: 30 von 41 kontaktierten neurologischen (N) und 12 von 33 psychiatrischen Einrichtungen (P) (insgesamt 57 %) beantworteten die Befragung. Die Anzahl von Erstuntersuchungen/Jahr von Personen (P) mit V. a. neurokognitive Störungen beträgt median in N 100 (0–313), in P 100 (0–500), davon haben in N 45 % (0–80) und in P 25 % (10–50) der P einen MMSE-Score von ≥ 22 Punkten (Einschlusskriterium). Kontrolluntersuchungen erfolgen in N in 57 (0–508) und in P in 40 (0–800) Fällen, vor Ort in 17,5 % (0–90) bzw. 10 % (0–95). Es wird von allen N und P in 100 % der P eine Anamneseerhebung durchgeführt, in jeweils 90 % Neurostatus, in 87 % bzw. 90 % Routine-Blutuntersuchung und ein kognitives Screening. Eine detaillierte neuropsychologische Testung erfolgt in N in median 70 % (10–100) der Fälle und in P in 50 % (15–80), zerebrales MRT in N in 90 (50–100), in P in 45 (10–95), CT in N in 27,5 % (0–80) und in P in 10 % (0–80), sowie Amyloid- und FDG-PET in N in 5 bzw. 15 % (0–70 bzw. 0–80) und in P in 5 bzw. 7,5 % (0–30). Liquor-Untersuchungen auf neurodegenerative Parameter (Aß42, Aß40, Tau, P-Tau), gegenüber Amyloid-PET bevorzugt in 16 von 37 Zentren, werden in N in 40 % (0–90) und in P in 7,5 % (0–30), genetische Untersuchungen (v. a. Apo-E-ε-Status) in 15 % (0–75) und 8 % (0–15) sowie EEG in 30 % (0–100) und 10 % (0–80) der Fälle durchgeführt. Zu einer Amyloid-Antikörper-Therapie bereit sind 13 von 25 N, 2 von 12 P; regelmäßige MRT-Kontrollen, v. a. in Kooperation mit niedergelassenen Radiolog*innen, können sich 16 von 30 N und 7 von 11 P vorstellen. Ressourcenmängel betreffen v. a. Ärzt*innen, Raumreserven, Psycholog*innen, Amyloid-PET und administratives Personal. Nahezu alle N und P befürworten dringend multidisziplinäre Versorgungsnetzwerke. Interesse an einem österreichweiten Demenzregister besteht bei 22 von 30 N und 6 von 12 P (Einschränkung: Aufwand!).

Zusammenfassung: Die Rücklaufquote von über 57 % erlaubt Rückschlüsse für Österreich. Kapazitäten und Ressourcen sind uneinheitlich, für den zu erwartenden Aufwand deutlich zu gering und bedürfen einer raschen Verbesserung in mehrfachen Bereichen unter Berücksichtigung des von der ÖAG errechneten Zeit/Personal-Aufwandes und Strukturkriterien. Wichtig sind Patienten-Pathways und der rasche Aufbau von Versorgungsnetzwerken.

V16: Voxel-basiertes Lesion Symptom Mapping zur Prädiktion von Poststroke-Epilepsie – Ergebnisse einer multizentrischen Studie

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Hintergrund: Die Poststroke-Epilepsie (PSE) ist eine bedeutende Langzeitkomplikation nach einem Schlaganfall. Daten zu Prädiktoren von PSE bei Patient*innen nach Schlaganfall durch eine Großgefäßokklusion, die mittels mechanischer Thrombektomie (MT) behandelt wurden, sind begrenzt. Voxel-basiertes Lesion Symptom Mapping auf Basis von MRT-Aufnahmen könnte ein wertvolles Instrument zur Risikobewertung von PSE darstellen. Ziel dieser Studie war es, das Risiko einer PSE nach akutem Schlaganfall, der mittels MT behandelt wurde, durch Voxel- und volumetrische Analysen zu untersuchen.

Fragestellung: 1. Hat das Läsionsvolumen einen Einfluss auf das Risiko einer PSE? 2. Gibt es spezifische Hirnareale, die das Risiko einer PSE erhöhen?

Methoden: In dieser bizentrischen Studie, durchgeführt an 2 tertiären Schlaganfallzentren, wurden konsekutive Patient*innen mit akutem ischämischem Schlaganfall eingeschlossen, die zwischen 2011 und 2017 eine MT erhalten hatten. Voraussetzung für die Teilnahme war das Vorliegen einer ▶

postinterventionellen zerebralen MRT sowie langfristiger Follow-up-Daten. Das Infarktvolumen und die Infarktlokalisation wurden auf den MRT-Bildern analysiert. Das Voxel-basierte Lesion Symptom Mapping basierte auf FLAIR-Scans (Fluid-Attenuated Inversion Recovery). Nach einer semiautomatisierten Umrisszeichnung der Läsionen und Erstellung binärisierter Läsionsmasken wurden alle FLAIR-Scans und Läsionsmasken auf eine Gruppenvorlage registriert. Das Voxel-basierte Lesion Symptom Mapping erfolgte mithilfe der Software NiiStat, um relevante topographische Läsionsmuster bei PSE zu identifizieren.

Ergebnisse: Von 348 analysierten Patient*innen mussten 95 aufgrund ungenauer Läsionsregistrierung ausgeschlossen werden. Letztendlich wurden die Läsionskarten von 251 Patient*innen (medianes Alter: 66 Jahre, 45,4 % Frauen) für das Voxel-basierte Lesion Symptom Mapping berücksichtigt, darunter 26 Patient*innen mit PSE (10,4 %). Das mittlere Infarktvolumen war bei Patient*innen mit PSE signifikant höher (119,2 cm^3 vs. 43,9 cm^3, $p < 0,0001$). Das Voxel-basierte Lesion Symptom Mapping identifizierte spezifische Hirnregionen, die mit PSE assoziiert waren: den Gyrus rectus, den orbitofrontalen Gyrus, die frontalen, parietalen und temporalen Opercula sowie den Temporallappenpol (Cortex piriformis).

Zusammenfassung: Neben dem Infarktvolumen identifizierte das Voxel-basierte Lesion Symptom Mapping auf postinterventionellen MRTs spezifische Hirnregionen, die mit PSE nach MT bei Schlaganfällen durch Großgefäßverschluss assoziiert sind. Diese Erkenntnisse könnten zur Risikostratifizierung und Nachsorge in dieser spezifischen Patientengruppe beitragen.

V17: Characteristics of young stroke patients with migraine regarding cardiovascular risk factors and stroke etiology: Results of the STROKE-CARD Trial

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Background: The association between migraine and ischemic stroke, particularly in younger individuals, is well established, yet the underlying mechanisms remain unclear.

Method: We present the results of the STROKE-CARD Trial long-term follow-up (Clinicaltrials.gov number NCT04205006)—a representative cohort of ischemic stroke patients—focusing on cardiovascular risk factors and stroke characteristics in young stroke patients ≤ 55 years of age with and without migraine. Headaches were classified based on the most recent ICHD-3 criteria and evaluated by headache specialists through structured face-to-face or telephone interviews.

Results: Of the 262 young stroke patients, 78 (27.7 %) had a history of migraine, with 45 (57.7 %) experiencing migraine without aura and 33 (42.3 %) with aura. Migraine prevalence was higher in women (55.1 %) than in men (44.9 %) ($p < 0.001$). The analysis revealed that migraine patients were less likely to have conditions such as arterial hypertension ($p = 0.045$), dyslipidaemia ($p = 0.016$), and smoking ($p = 0.020$). Migraine was independently associated with a younger age at stroke onset ($p = 0.040$; OR 2.06, 95% CI [1.03–4.12]). At hospital discharge, NIHSS and mRS scores were significantly lower in migraine patients compared to non-migraineurs ($p = 0.001$).

Conclusion: Migraine in young stroke patients is associated with a lower prevalence of traditional cardiovascular risk factors and an earlier age of stroke onset. The importance of early, more extensive screening and targeted stroke prevention strategies in young migraine patients is highlighted by these findings.

V18: Neurosurgical interventions in idiopathic intracranial hypertension: A comprehensive multicenter study of outcome and referral pattern

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Background: Neurosurgical interventions are recommended for fulminant or treatment-refractory idiopathic intracranial hypertension (IIH). However, evidence on their outcomes remains limited, particularly regarding the impact of referral patterns and indications.

Aim: This study aimed to evaluate the clinical outcomes and referral patterns for neurosurgical interventions in IIH, identifying predictors of beneficial or adverse outcomes.

Method: A retrospective multicenter study was conducted by the Danish-Austrian IIH Consortium (DASH-IIH). Databases from 3 centers (Vienna, Odense, Copenhagen) were screened for patients with IIH (pwIIH) meeting the revised Friedman criteria who underwent neurosurgical intervention between April 1 2014 and April 30 2024 with at least 6 months of documented follow-up. Outcomes assessed at 6 months post-intervention (M6) included visual function (visual acuity and/or visual fields), headache frequency (monthly headache days [MHD]), papilledema resolution, and severe adverse events (CTCAE grade ≥ 3). Multivariate regression models were applied to adjust for confounders.

Results: Thirty-six pwIIH were included (100 % female, mean age: 32.5 years, median BMI: 37.0, median CSF opening pressure: 41 cmH_2O). Of these, 27 (75 %) underwent ventriculo-peritoneal shunting (VPS) and 9 (25 %) underwent optic nerve sheath fenestration (ONSF). The primary indication was acute or imminent visual loss in 30 patients (83.3 %) and refractory headache in 6 (16.7 %).

Visual function improved in 41.7 % of patients, with papilledema resolving in 89.7 %. A clinically relevant reduction in headache frequency (i.e. ≥ 50 %) was observed in 30.6 % of patients, with a median reduction of 4.5 MHD.

Multivariate analysis showed no significant differences between VPS and ONSF in terms of visual or headache outcomes, nor in the rate of severe adverse events. Notably, referrals for refractory headache were not associated with visual improvement (0 % [0/6]) and were significantly less likely to result in headache improvement (odds ratio 0.11, p = 0.012).

Conclusion: VPS and ONSF are effective interventions for acute or imminent visual loss in IIH, significantly improving visual function and headache outcomes. However, refractory headache alone appears to be an inappropriate indication for neurosurgical referral.

P01: Visuelle und verbale Gedächtnis-Konnektivität bei Temporallappenepilepsie

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Die Temporallappenepilepsie (TLE) wird zunehmend als Netzwerkerkrankung angesehen. Von der Epilepsie betroffene Netzwerke gehen oft weit über den Anfallsursprung hinaus. Neben epileptischen Anfällen kommt es zusätzlich durch die Beeinträchtigung kognitiver Funktionen wie der Sprach- und Gedächtnisleistung zu einer relevanten Einschränkung der Lebensqualität der Patient*innen. Bei etwa 70 % der operativ behandelten therapierefraktären TLE-Patient*innen kann zwar eine Anfallsfreiheit erreicht werden, jedoch führen diese operativen Eingriffe häufig zu einer weiteren Zunahme der kognitiven Beeinträchtigung. Mithilfe funktioneller Konnektivitätsanalysen können involvierte funktionelle Netzwerke dargestellt werden. Ziel dieser Studie war es, mithilfe von Konnektomanalysen die Organisation visueller und verbaler Gedächtnisnetzwerke in links- und rechtsseitigen TLE-Patient*innen darzustellen. Weiters sollten durch Korrelation mit entsprechenden neuropsychologischen Testergebnissen effektive verbale und visuelle Gedächtnisnetzwerke sowie potenziell prädiktive netzwerkbasierte Muster und Marker für verbale und visuelle Gedächtnisleistung identifiziert werden. Es wurden insgesamt 61 Patient*innen mit TLE (35 links, 26 rechts) eingeschlossen. Im Rahmen der prächirurgischen Abklärung erhielten alle Patient*innen auch eine ausführliche neuropsychologische Testung sowie eine funktionelle Magnetresonanztomographie (fMRT). An einem 3 Tesla MRT Scanner führten die Patient*innen neben Sprachparadigmen auch ein Gedächtnisparadigma durch, mit dem es möglich war, verbales und visuelles Gedächtnis in 1 Sitzung zu untersuchen. Während der 12-minütigen fMRT-Messung wurden den Patient*innen über einen Bildschirm unterschiedliche Stimuli (Gesichter, Bilder von Objekten und Wörter) präsentiert. Im Anschluss an die Messung fand ein Erinnerungstest außerhalb des Scanners statt, dessen Ergebnisse für die Event-related Analyse verwendet wurden. Die Daten wurden mit fMRIprep präprozessiert und in den Programmen Statistical Parametric Mapping 12 (SPM12) und CONN-Toolbox analysiert. Für die Korrelation der funktionellen Konnektivität während der visuellen und verbalen Gedächtnisaufgaben und den neuropsychologischen Testergebnissen wurde eine multiple Regressionsanalyse durchgeführt. Präliminäre Ergebnisse zeigten signifikante Unterschiede in den verbalen und visuellen Gedächtnisnetzwerken zwischen links- und rechtshemisphärischen TLE-Patient*innen. Die Konnektitivät der verbalen Gedächtnisnetzwerke zeigte sich insbesondere bei den linksseitigen TLE-Patient*innen beeinträchtigt, während bei den rechtsseitigen eher die Konnektivität der visuellen Gedächtnisnetzwerke beeinträchtigt war. Korrelationsanalysen mit den neuropsychologischen Testergebnissen zeigten, dass bei rechts- und linksseitigen TLE-Patient*innen eine höhere Konnektivität des jeweiligen ipsilateralen Hippocampus zur kontralateralen Hemisphäre mit einer besseren verbalen und visuellen Gedächtnisleistung assoziiert war. Mittels Konnektivitätsanalysen können Unterschiede in visuellen und verbalen Gedächtnisnetzwerken bei TLE-Patient*innen charakterisiert und vulnerable Regionen und Verbindungen vor potenziellen Temporallappenresektionen identifiziert werden. Die Reorganisation dieser Netzwerke im Rahmen einer Epilepsieerkrankung betrifft oft Areale, die weit über den Anfallsursprung hinausreichen. Mit einem größeren Verständnis dieser Gedächtnisnetzwerke sollen Schlüsselregionen identifiziert werden, um bei TLE-Patient*innen sowohl prä- als auch postoperativ kognitive Beeinträchtigungen besser vorhersagen und letztlich minimieren zu können.

P02: Simultane Stereo-EEG- und Skalp-EEG-Analyse zur präzisen Eingrenzung epileptogener Zonen bei MRT-negativer Temporallappen-Epilepsie

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Hintergrund: In etwa 40 % der pharmakoresistenten fokalen Epilepsien fehlt ein strukturelles MRT-Korrelat. Zur präzisen Abgrenzung der epileptogenen Zone (EZ) wird ein invasives Elektroenzephalogramm (EEG) mittels Stereo-EEG-Elektroden angewendet. Eine simultane Aufzeichnung von Skalp-EEG und SEEG ermöglicht eine differenzierte Analyse iktaler und interiktaler Entladungen und trägt zur Optimierung invasiver Diagnostik und therapeutischer Strategien bei.

Fragestellung: Ziel der Studie ist es, bei MRT-negativer Temporallappen-Epilepsie (TLE) die interiktale und iktale epilepsietypische Potenziale (ETPs) an invasiven Elektrodenkontakten zu identifizieren und deren Eigenschaften mit den simultan registrierten Skalp-EEG-Signalen zu vergleichen.

Methode: Alle erwachsenen Patient*innen mit MRT-negativer therapieresistenter TLE, die zwischen 2016 und 2023 an der Universitätsklinik für Neurologie einer simultanen Skalp- und SEEG-Untersuchung unterzogen wurden, wurden in die Analyse retrospektiv eingeschlossen. Die Studie wurde von der zuständigen Ethikkommission der Medizinischen Universität Wien genehmigt (Ethikvotum-Nummer: 2161/2024). Die Anfälle wurden anhand der zeitlichen Latenz zwischen SEEG- und Skalp-EEG-Beginn in 3 Gruppen eingeteilt: Gruppe 1: ≤ 10 s, Gruppe 2: > 10 s, Gruppe 3: keine Skalp-EEG-Aktivität.
Das erste relevante iktale Muster (Seizure Onset Pattern [SOP]) im SEEG wurde in iktale paroxysmale schnelle Aktivität (iPSA), hochamplitudige Polyspikes (HAP) und rhythmische Sharp Waves (rSW) eingeteilt. SOP im Skalp-EEG wurde in Abflachung und rhythmischer Deltaaktivität (rDA) eingeteilt. Interiktale ETPs (Sharp Waves [SW] und interiktale rhythmische Deltaaktivität [iiRDA]) wurden nach Lokalisation, Morphologie und Amplitude zu unterschiedlichen Spike-Populationen zugeordnet.

Ergebnisse: 10 Patient*innen (alle männlich, medianes Alter 28 [20–45] Jahre) erfüllten die Einschlusskriterien. Es wurden 42 Anfälle analysiert, davon wurden 22 (52,4 %) der Gruppe 1, 11 (26,2 %) der Gruppe 2 und 9 (21,4 %) der Gruppe 3 zugeordnet. SEEG-SOP in Gruppe 1 zeigten überwiegend iPSA und HAP (je 40,9 %), in Gruppe 2 primär HAP (45,4 %), gefolgt von iPSA und rSW (je 27,3 %), und in Gruppe 3 kamen HAP und rSW gleichermaßen vor (je 44,4 %). Die SOP wurde in Gruppe 1 zu 50 %, in Gruppe 2 zu 100 % und in Gruppe 3 zu 55,6 % an distalen Kontakten detektiert.Im Skalp-EEG zeigten die Anfälle der Gruppe 1 eine Abflachung (50 %) oder rDA (45,4 %) und in Gruppe 2 überwiegend rDA (63,6 %). In Gruppe 1 waren Abflachung und rDA regional (90,1 % resp. 80 %), und rDA in Gruppe 2 nicht lateralisiert (71,4 %). Insgesamt wurden in SEEG 38 Populationen interiktaler ETPs detektiert, davon waren 19 (50 %) im Skalp-EEG

sichtbar. Lediglich in 9,8 % (Spanne 0,0–38,4 %) der im SEEG registrierten ETPs wurden auf dem Skalp detektiert. Im Skalp-EEG sichtbare ETPs fanden sich häufiger an proximalen SEEG-Kontakten, während tief, distal erfasste ETPs meist kein Skalp-EEG-Korrelat zeigten (81,8 %). **Zusammenfassung:** Tiefe epileptische Entladungen bei MRT-negativer TLE bleiben häufig auf das SEEG beschränkt, da Morphologie und Lokalisation die Detektierbarkeit am Skalp limitierten. Eine Korrelation beider Verfahren ermöglicht die präzisere Eingrenzung der EZ und optimiert das therapeutische Vorgehen.

P03: Therapeutic interventions and outcomes

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Background: Anti-IgLON5 disease is a rare and complex neurological disorder characterized by a wide clinical spectrum. Effective management remains challenging due to the disease's heterogeneity and unpredictable outcomes.
Aim: This systematic review and meta-analysis aimed to evaluate the therapeutic interventions and reported outcomes in patients with anti-IgLON5 disease.
Method: A systematic search was conducted using PubMed/MEDLINE, Web of Science, and Semantic Scholar for case reports and case series on anti-IgLON5 disease. English-language publications reporting patients with positive IgLON5 antibodies in serum or cerebrospinal fluid (CSF) were included. Data on therapeutic interventions and outcomes were extracted and analyzed.
Results: Various immune-targeted treatments were administered to 46 patients. Steroids were the most common treatment, used in 37 patients, followed by high-dose methylprednisolone (24 patients), intravenous immunoglobulin (23 patients), and other therapies such as rituximab and plasma exchange. Patient outcomes varied: 25 patients showed improvement, 6 remained stable, and 7 worsened. Five patients initially improved but later passed away, with a total of 8 deaths reported. The reported causes of death included unknown reasons (6 cases), sleep-related deaths (2 cases), infection (1 case), and cardiac arrest 7 years after initial presentation (1 case). In 8 cases, outcomes were not reported.
Conclusion: Immune-targeted therapies, particularly steroids and intravenous immunoglobulin, have been used most often in anti-IgLON5 disease. While many patients show improvement, the disease remains associated with significant morbidity and mortality.

P04: Anti-IgLON5 disease: A systematic review and meta-analysis: Therapeutic interventions and outcomes

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Background: Anti-IgLON5 disease, first identified in 2014, is a complex neurological disorder with heterogeneous sleep, movement, neuroimmunological, and neurodegenerative features. Its clinical complexity creates challenges in establishing an early diagnosis.
Aim: This systematic review and meta-analysis aimed to characterize the laboratory findings associated with anti-IgLON5 disease to improve diagnostic precision.
Method: A systematic search was conducted using PubMed/MEDLINE, Web of Science, and Semantic Scholar for case reports and case series on anti-IgLON5 disease. Inclusion criteria were English-language publications reporting patients with positive IgLON5 antibodies in serum or cerebrospinal fluid (CSF). Case series with ≥ 10 patients were included in the meta-analysis, while all other reports were included in the systematic review.
Results: The analysis included 169 patients from large case series (n ≥ 10) and 91 patients from smaller case reports and series (n < 10). IgLON5 antibodies were

detected in up to 98 % of cases through serum testing, with only 1 case showing positivity exclusively in CSF. Routine CSF analysis results were nonspecific, with elevated white blood cell counts reported in 36 %, elevated CSF total protein in 58 %, abnormal glucose levels in 0 %, and oligoclonal bands in 21.4 % of cases. Genetic testing revealed a high prevalence of HLA-DQB105:01 (83.3 %) and HLA-DQB110:01 (58.3 %).

Conclusion: Laboratory findings indicate that serum antibody testing for IgLON5 has high sensitivity, reducing the need for CSF testing in most cases. Routine CSF analysis is often inconclusive, and genetic markers such as HLA-DQB1*05:01 may support diagnosis. These findings provide valuable diagnostic insights, emphasizing the importance of serum antibody testing in suspected cases.

P05: OCT improves risk stratification for PIRA at diagnosis of relapsing multiple sclerosis

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Background: Progression independent of relapse activity (PIRA) is a main driver of disability accumulation in relapsing multiple sclerosis (RMS). Given the availability of disease-modifying treatments (DMT) with differing degree of efficacy and risk, biomarkers enabling early prognostic stratification are urgently required. Retinal layer thickness measured by optical coherence tomography (OCT) reflects neuroaxonal damage, an important pathophysiological mechanism underlying PIRA.

Objective: To investigate whether assessment of retinal layer thickness at RMS diagnosis improves prediction of PIRA.

Method: From a prospective observational study, we included patients with newly diagnosed RMS and an OCT scan within 90 days after RMS diagnosis, excluding eyes with optic neuritis. Impact of peripapillary retinal nerve fiber layer (pRNFL) and macular ganglion-cell-and-inner-plexiform-layer (GCIPL) thickness for prediction of PIRA (defined as EDSS increase ≥ 1.5/1.0/0.5 from baseline 0/1-4.0/≥ 4.5 ≥ 90 days from relapse) was investigated by multivariate (adjusted hazard ratio [HR] with 95% confidence intervals [CI]) Cox regression models adjusting for clinical and MRI covariates (T2 lesion load, presence of infratentorial lesions).

Results: We analyzed 313 RMS patients (mean age 30.5 years [SD 7.9], 74.1 % female) over a median observation period of 70 months (range: 12–105). Mean pRNFL and GCIPL thickness were 92.2µm (11.8) and 81.1 µm (10.9), respectively. Ninety-seven (31 %) patients received highly effective DMT (HE-DMT) and 190 (60.7 %) moderately effective DMT. PIRA occurred in 76 (24.3 %) patients after a median 47 months (6–99). While higher age, incomplete remission of first relapse, and infratentorial MRI lesions were all associated with higher risk of PIRA, first-line use of HE-DMT significantly decreased the risk of PIRA (HR 0.69 [CI 0.41–0.97], p = 0.027). Both lower pRNFL (HR 1.6 per 5µm, [CI 1.1–1.8], p < 0.001) and GCIPL thickness at diagnosis (HR 1.7 per 5µm, [CI 1.3–3.1], p < 0.001) were independent predictors of PIRA. Adding retinal thickness to the multivariate model improved predictive accuracy for PIRA from 48 % to 62 % (p < 0.001).

Interpretation: Retinal layer thickness assessed by OCT improved stratification for risk of PIRA at RMS diagnosis, likely identifying patients with already pronounced subclinical neuroaxonal damage and potentially informing treatment strategy.

P06: Stratifying the risk of disease reactivation after DMT de-escalation/ discontinuation in relapsing multiple sclerosis by the VIAADISC score

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Background: Evidence regarding de-escalation of disease-modifying therapy (DMT) in relapsing multiple sclerosis (RMS) is scarce. The Vienna Innsbruck score based on age, MRI activity, and duration in stable course (VIAADISC) was shown to predict risk of disease reactivation after discontinuation of interferon beta and glatiramer acetate (BRACE).

Objective: To investigate whether the VIAADISC score can be used to predict disease reactivation in RMS patients after de-escalation/discontinuation.

Method: Aggregating data from 5 MS centers in Austria and Switzerland, we included RMS patients who i) continuously received any DMT other than BRACE for ≥ 12 months; ii) were de-escalated (i.e. switched from high-efficacy DMT [H-DMT] to moderate-efficacy DMT [M-DMT]) or discontinued M-DMT for ≥ 6 months); iii) had a brain MRI at de-escalation; and iv) had ≥ 2 years of follow-up. The primary endpoint was reccurrence of clinical disease activity (relapse and/or EDSS progression) during follow-up. VIAADISC score (0–6; age < 45/45–54/≥ 55 = 2/1/0 points, MRI activity = 2 points, duration without clinical disease activity < 4/4–8/> 8 years = 2/1/0 points) was calculated. Cox regression was employed to determine predictive value of VIAADISC adjusting for covariates including DMT substances.

Results: Of 129 RMS patients included (65.1 % female), 41.9 % discontinued M-DMT (27.1 % dimethylfumarate [DMF], 9.3 % teriflunomide [TERI]), and 58.1 % were de-escalated from H-DMT (44.2 % natalizumab [NTZ], 14.0 % fingolimod [FTY], 5.5 % rituximab [RTX]) at a median age of 44 years (IQR 34–53) and a median disease duration of 14 years (IQR 7–21). At de-escalation, median duration without disease activity was 2.4 years [1.5–3.9] and 93.1 % were without MRI activity, resulting in a median VIAADISC score of 3 (IQR 2–4, range 0–6). Observed over a median 6.0 years (3.5–8.7), clinical disease activity reoccurred in 56.6 % overall, most frequently when de-escalating from NTZ (70.2 %) and FTY (66.7 %). In Cox regression, higher VIAADISC scores independently predicted an increased risk of recurrent disease activity (HR 1.71 per point [95 % CI 1.31–2.12], p = 0.012), as did de-escalation from NTZ/FTY (HR 2.54 [CI 1.09-5.91], p = 0.031). Patients on DMF/TERI with a VIAADISC score ≤ 1 had only a 4.8 % risk of recurrent disease activity.

Conclusion: Risk of disease reactivation after DMT de-escalation can be stratified based on age, MRI activity, and duration in stable course by the VIAADISC score, which may support patients and neurologists in the process of decision making to de-escalate/discontinue DMTs. De-escalation from NTZ/FTY is associated with increased risk and should be avoided by lateral switch.

P07: The rs10191329 risk allele is associated with retinal layer thinning in multiple sclerosis

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Background: The minor allele of the genetic variant rs10191329 in the DYSF–ZNF638 locus, which is implicated in CNS resilience rather than immune-mediated pathology, has been associated with faster disability progression in

multiple sclerosis (MS). Retinal layer thinning measured by optical coherence tomography (OCT) is an established biomarker of neuroaxonal damage.

Objective: To investigate whether the rs10191329 variant is associated with retinal layer thinning in relapsing MS.

Method: From a prospective observational study, we included patients with RMS and ≥ 2 OCT scans excluding eyes with optic neuritis during the observation period. DNA samples were genotyped on an Illumina Infinium Global Screening Array-24 (GSA v3.0+MD), imputing individual variants onto the Haplotype Reference Consortium panel (Release 1.1) using Minimac4 (V1.0.2). We used a multivariate linear regression model with mean annualized rates of retinal layer thinning (%/year) in peripapillary retinal nerve fiber layer (aLpRNFL), macular ganglion-cell-and-inner-plexiform-layer (aLGCIPL) thinning as the dependent variable, and rs10191329 risk allele number (rs10191329*A) as the independent variable, adjusting for age, sex, and 10 ancestry components.

Results: We included 208 RMS patients (mean age: 30.5 years [SD 7.9], 74.1 % female, median EDSS: 2.0 [range 0–6.5], median observation period: 25 months [range 12–105], median number of OCT scans: 3 [2–6]). We found that the risk allele of rs10191329 was associated with higher rates of retinal thinning (estimate 0.119 [standard error 0.056], one-sided $p < 0.001$). Each rs10191329*A allele was associated with an increase of aLpRNFL by 0.10%/year (95 % confidence interval [CI] 0.05–0.19, $p < 0.001$) and aLGCIPL by 0.11 %/year (95 % confidence interval [CI] 0.07–0.19, $p < 0.001$), corresponding to 26.4 % (CI 13.1–44.2) and 27.2 % (CI 14.3–43.1) of mean atrophy rates (aLpRNFL 0.42%/y [0.31–0.59], aLGCIPL 0.21%/y [0.15-0.29]).

Interpretation: Carriers of the minor allele of rs10191329 seem more prone to MS-related tissue damage. While clinical implications are currently unclear, stratification for this genotype in clinical trials may be reasonable.

P08: Application of optical coherence tomography in multiple sclerosis: consensus recommendations of the Austrian network (AN-OCT-MS)

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Background: Optical coherence tomography (OCT) for measuring retinal layer thicknesses is a new modality relevant for diagnosis and prognosis of multiple sclerosis (MS), now anchored in clinical routine with the 2024 revision of the McDonald criteria.

Objective: The aim of this consensus is to provide a comprehensive recommendation for the use of OCT in patients with MS (pwMS).

Method: This consensus was developed by an expert committee (Austrian Network for Optical Coherence Tomography in Multiple Sclerosis [AN-OCT-MS]) set up under the auspices of the Austrian Society of Neurology and the Austrian Society of Ophthalmology following a formal consensus methodology. It covers indications as well as quality standards for conduction, interpretation, and reporting of OCT in pwMS.

Results: All 20 recommendations achieved a strong consensus (> 90 %) including: OCT should be performed as part of diagnostic workup for suspected MS. If a pathological inter-eye-difference (IED) is detected in peripapillary retinal nerve fiber layer (pRNFL, IED ≥ 5µm/≥ 5 %) and/or ganglion-cell-and-inner-plexiform-layer (GCIPL, IED ≥ 4µm/≥ 4 %), involvement of the optic nerve for fulfillment of dissemination in space (DIS) is considered proven, provided other causes have been excluded.

OCT should be performed at diagnosis or before treatment decisions to stratify for progression risk: If there are signs of advanced neuroaxonal damage (pRNFL ≤ 88µm and/or GCIPL< 77µm), an increased risk of disability progression over the next 3 years can be assumed, provided other causes have been excluded.

The use of OCT in DMT monitoring cannot currently be generally recommended. At centers particularly familiar with OCT in MS, it may be used to monitor MS-associated neuroaxonal damage, whereby results should only be interpreted in the overall clinical context and never used as the sole decision criterion.

OCT scans should include a peripapillary ring scan and a multilinear macular scan according to AN-OCT-MS protocol.

OCT scans should undergo quality control based on the OSCAR-IB criteria and only be used if quality criteria are met.

Only Spectralis® (Heidelberg Engineering) or Cirrus® (Carl Zeiss Meditec) devices should be used in MS patients, as other devices are not currently validated in MS.

The report on findings of an OCT in pwMS should contain a summarizing interpretation.

Conclusion: The AN-OCT-MS guidelines provide a comprehensive framework to facilitate the use of OCT in patients with MS. A growing network of centers following the AN-OCT-MS consensus provides the basis for comprehensive nationwide high-quality OCT for pwMS in Austria.

P09: Dissecting disability increase in people with MS: RAW vs. PIRA in a real-world cohort

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Background: The distinction of relapse associated disability worsening (RAW) and progression occurring independent of relapses (PIRA) has significantly changed the field's view on accumulation of disability in relapsing multiple sclerosis (RMS). The Expanded Disability Status Scale (EDSS) is the most widely used measure for disability worsening or treatment response, with time to EDSS worsening serving as a common endpoint.

Problem: The large number of definitions for EDSS progression and for attributing progression to RAW or PIRA available in the literature, combined with incomplete reporting of definition details, hampers comparability and interpretation of study results and may create confusion in clinical practice. Although harmonization proposals and the corresponding algorithms have been published recently, they are tailored to well-structured clinical trial data. A systematic analysis of the extent to which varying definitions of disability worsening, RAW, and PIRA affect their frequency and/or time to event in patients with relapsing MS in a large, representative real-world cohort is still lacking.

Method: We analyzed event rates, event type distributions, and the contribution of each event type to the total disability accumulation in from the Austrian MS Treatment Registry (AMSTR) for 1440 definitions of EDSS progression using a newly developed algorithm specifically tailored to retrospective registry data. Included were follow-ups from RMS patients ≥ 18 years old with a minimum duration of 24 months, at least 3 EDSS assessments overall, and at least 1 EDSS assessment every 12 months.

Results: In 3525 follow-up periods analyzed, 1842 (52.3 %) had no documented relapse and 1361 (38.6 %) follow-ups had at least 1 documented relapse during the follow-up period. Overall, between 712 and 2352 (median 1133) progression events of any type and a total EDSS score increase from 856 to 3035 (median 1388) points were observed. Event rates (fraction of follow-ups with at least 1 progression event irrespective of type) ranged from 16.1 % to 41.6 % with a mean event rate of 25.7 %.

The contribution of PIRA to the total number of EDSS progression events ranged from 61.5 % to 88.8 % (mean 73.3 %), and the contribution of RAW ranged from 5.0 % to 24.1 % (mean 12.4 %). Between 60.6 % and 88.6 % (mean 72.4 %) of the total EDSS increase was due to PIRA, 4.9 % to 25.9 % (mean 12.9 %) was due to RAW. 0 % to 19.7 % (mean 7.9 %) of all events and 0 % to 20.2 % (mean 8.2 %) of the total EDSS increase did not meet the requirements for RAW or PIRA and were thus labeled as "undefined." The algorithm developed for this analysis will be made publicly available.

Summary: PIRA is the main driver of disability accumulation even in patients with RMS. However, the definition of disability progression strongly impacts event rates and event type, underlining the importance of detailed reporting of definitions used in studies. Our algorithm provides a framework for reproducible analysis of real-world data.

P10: Age-related dynamics of glial fibrillary acidic protein blood levels in a normal aging cohort: Implications for biomarker studies in neurological diseases

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Introduction: Glial fibrillary acidic protein (GFAP) is an astrocytic biomarker that is upregulated in various neurological conditions, including multiple sclerosis (MS). In MS, higher serum GFAP (sGFAP) are associated with disability worsening and MS-related magnetic resonance imaging (MRI) changes. However, for correct interpretation of sGFAP, detailed knowledge of its variability with age and its temporal dynamics in neurologically incons- ▶

picuous individuals is crucial. This has not yet been investigated.

Objective: To assess age-related temporal dynamics and sex differences of sGFAP in a large, longitudinal normal aging cohort.

Method: 316 (mean age: 64.5 ± 10.7 years, range: 38–82, 184 female) neurologically inconspicuous individuals participating in a community-dwelling cohort study (ASPS-Fam), with longitudinal data (mean follow-up duration: 5.6 ± 1.0 years) available from 89 participants, were included in this study. All participants underwent comprehensive diagnostic work-up including a detailed neurological examination, 3T brain-MRI, cognitive, and laboratory evaluation. sGFAP was measured using a single molecule array (Simoa HD-X).

Results: sGFAP significantly increases with age ($r = 0.5$, $p < 0.001$) (< 50 years [pg/mL, mean ± SD] [73.1 ± 25.4], 50–60 years [86.8 ± 35.1], 60–70 years [136.9 ± 48.4], > 70 years [154.6 ± 60.7]) and tendentially higher sGFAP levels are found in females compared to males ($p = 0.05$). The increase of sGFAP with age is accompanied by an increase in the variability of this marker in the older age groups ($p < 0.05$). Longitudinal analyses showed a significant difference between males and females ($p = 0.02$), with a larger sGFAP increase in females.

Conclusion: sGFAP levels increase with age, which is accompanied by a higher variability of this marker in older individuals. This high variability of sGFAP in neurologically normal individuals needs to be taken into account when interpreting this marker in neurological disorders, and requires the establishment of normative values, e.g., based on percentiles or z-scores. Analyses of potential relationships between sGFAP, brain-MRI, cognitive measures, and other confounding factors are currently ongoing.

P11: Link between ovarian aging and multiple sclerosis: Anti-Müllerian hormone as a predictor of disease activity and disability worsening.

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Background: Ovarian aging as assessed by Anti-Müllerian hormone (AMH) has been implicated in various health outcomes in women. In multiple sclerosis (MS), the effect of aging on the disease course is well known. However, the role of ovarian aging in MS disease activity has not yet been studied.

Objective: To investigate the relationship between AMH levels and relapse rate as well as disability progression.

Method: This prospective longitudinal study including women with MS was conducted at the Medical University of Innsbruck. AMH levels measured by electrochemiluminescence Immunoassay were used as a biomarker of ovarian aging. Cox regression analysis was used to investigate the impact of AMH on time to relapse and time to disability progression, defined by an increase in EDSS (for a baseline EDSS of 0, an increase of ≥ 1.5; for a baseline EDSS between 1 and 5, an increase of ≥ 1.0; and for a baseline EDSS of ≥ 5.5, an increase of 0.5, confirmed after 6 months).

Results: A total of 104 women at the median age of 50 years, a median EDSS of 2.0, and a follow-up time of 48 months (IQR 43–50) were included. Univariate analyses revealed that women with relapses were younger (39 vs. 52 years), but also showed higher AMH levels (median 0.5 vs. 0 µg/l). Multivariable Cox regression analysis revealed that both age (HR 0.95, $p = 0.039$) and AMH levels (HR 1.40, $p = 0.022$) independently predicted time to relapse. With regard to disability progression, in univariate analysis, women were older (53 vs. 48) and had lower AMH levels (0 vs. 0.1 µg/l). Multivariable Cox regression analysis showed that the AMH level (HR 0.54, $p = 0.0038$) independent of age predicted a shorter time to disability progression.

Conclusion: In this study, we showed for the first time that ovarian aging, as reflected by AMH, is a risk factor in the clinical course of MS in addition to chronological age. These findings suggest that AMH may serve as an additional biomarker for predicting relapses and progression in women with MS.

P12: Expanded monitoring of patients with MS: Insights from the Vienna MS-nurse pilot project in clinical practice

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Introduction: Continuous monitoring of clinical performance metrics in patients with multiple sclerosis (pwMS) is critical for quantifying disability and detecting subtle changes in disease progression. Key measures such as the Symbol Digit Modalities Test (SDMT), Nine-Hole Peg Test (9HPT), Timed 25-Foot Walk (T25FW), as well as patient-reported outcomes (e.g., Fatigue Scale for Motor and Cognitive Functions [FSMC], the Hospital Anxiety and Depression Scale [HADS], and the Multiple Sclerosis Impact Scale [MSIS-29]) provide comprehensive insight into cognitive, motor, and emotional functioning. However, time constraints often limit the systematic implementation of such tests in clinical practice. To address this limitation, we employed a specialized MS nurse to conduct expanded clinical monitoring in pwMS routinely.

Objective: To determine the relevance of routine expanded clinical assessments performed by an MS nurse in a cross-sectional cohort of pwMS.

Method: The Department of Neurology at the Vienna General Hospital allocated personnel resources to employ a specialized MS nurse to administer clinical measures and questionnaires—including SDMT, 9HPT, and T25FW alongside FSMC, HADS, and MSIS-29—by referral by their treating neurologists following a standard operating procedure. Data from the assessments was descriptively analyzed, with abnormal results defined according to normative standards.

Results: Seventy-three pwMS (mean age: 33.6 [SD ± 12.7] years, 62 % female) were analyzed. Most patients were diagnosed with RRMS (69.4 %) and had a median EDSS of 3.0 (IQR 0–4.75) at the date of monitoring. SDMT was performed in 90 %, 9HPT in 85 %, and T25FW in 89 % of patients with a mean SDMT Z-Score of -0.44 (SD ± 1.4), median T25FW of 5.3 seconds (IQR 4.3–8.0), and a median 9HPT of 22.9 seconds (IQR 19.5–27.1m, averaged across hands). Abnormal results were reported in 23/65 (35 %) for SDMT, 26/62 (42 %) for 9HPT and 25/64 for T25FW of patients. Among patients with minimal clinical disability (EDSS ≤ 4), no reported neurologic deficit of the upper extremity, and no reported fatigue, the respective clinical measures were still frequently found to be abnormal (in these subgroups for T25FW 7/40 [18 %], 9HPT 8/37 [22 %] and SDMT 12/42 [29 %]). In patients with more severe disability or subjective cognitive impairment, rates of abnormal results were consistently high (for T25FW 21/24 [88 %], 9HPT 18/25 [72 %] and SDMT 11/24 [46 %]). Questionnaires were performed in more than half of patients (FSMC 55/72 [75 %], HADS 53/72 [73 %] and MSIS-29 37/73 [51 %]). Notably, FSMC and HADS were often moderately to severely abnormal in patients without reporting symptoms of fatigue or depression prior to testing (11/31 [36 %] for FSMC and 15/34 [44 %]).

Conclusion: Routine expanded clinical monitoring by an MS nurse frequently identified deficits across various domains, notably adding to deficits identified by routine history and EDSS rating in clinical practice. These findings underscore the importance of expanding routine testing and its potential to uncover subclinical impairments. Employment of specialized MS-nurses offers an effective strategy to address this unmet need while alleviating burden on treating physicians in standard clinical practice.

P13: Measuring comorbidity in multiple sclerosis: A systemic review of current comorbidity scales

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Introduction: Comorbidity assessment in multiple sclerosis (MS) is increasingly recognized as an important component due to its influence on treatment selection and clinical outcomes such as disability progression and quality of life. Yet, there is limited data on how to assess comorbidities in MS comprehensively and systematically. Despite the availability of several indices validated in other disease-specific populations, their application and validity in MS popu- ▶

lations remain unclear.

Objective: This study aimed to identify and evaluate comorbidity scales used in MS populations and to determine whether these indices are suitable for implementation in clinical practice by assessing their quality of evidence.

Method: A systematic review was conducted following predefined eligibility criteria. Studies published between 2014 and 2024 that reported original data or systematic reviews on measures of comorbidity in patients with multiple sclerosis (pwMS) were included. Studies solely involving healthy individuals, patients with other diseases, or measures unrelated to comorbidity were excluded. Information sources included MEDLINE, EMBASE, and the Cochrane Library. The search strategy combined the terms "multiple sclerosis" with "comorbidity." Quality of evidence was assessed by 4 raters using the GRADE approach.

Results: A total of 3178 studies were screened, of which 31 studies met inclusion criteria. These studies collectively analyzed data from 234089 patients with MS, predominantly using retrospective designs and nationwide registry databases. Four comorbidity scales were identified: the Charlson Comorbidity Index (CCI), Elixhauser Comorbidity Index (ECI), Self-Administered Comorbidity Questionnaire (SCQ), and Self-Reported Comorbidity Questionnaire for MS (SRQ-MS).

The most applied comorbidity scale was the CCI (14 studies, total sample size of 180001). Patients with higher EDSS scores (≥ 4.0) had greater comorbidity burdens (OR = 2.2), an increased mortality risk with CCI scores ≥ 4 (e.g., CCI ≥ 5, OR = 3.26, 95% CI: 1.58–6.70), and an increased risk of reaching disability milestones from CCI ≥1 (HR 1.23–1.62, $p < 0.001$). However, the quality of evidence for CCI in MS was graded as moderate to low due to limited direct validation and a predominant reliance on retrospective data, introducing a high risk of bias. Both the ECI (6 retrospective cohort studies) and SCQ (2 cross-sectional surveys and 1 longitudinal survey) demonstrated a notable absence of direct validation, as the analyzed studies did not define a clear and clinically meaningful outcome parameter. Furthermore, the reliance on retrospective data and analysis of cross-sectional survey data introduced a substantial risk of bias and was therefore graded with very low quality of evidence. Lastly, although the SRQ-MS (5 prospective, cross-sectional studies and 3 prospective, longitudinal cohort studies) was specifically validated in an MS population, its quality of evidence was determined to be moderate due to differences in study designs, lack of consistent follow-up, and potential indirectness due to the usage of modified SRQ-MS versions in some studies.

Conclusion: No single scale to determine comorbidities has been sufficiently validated for routine use in MS populations. The current scales, while potentially useful for research purposes, require further validation and standardization to ensure their applicability in real-world use. Future studies should focus on developing and validating MS-specific comorbidity scales.

P14: Measuring frailty in multiple sclerosis: A systemic review of current frailty indices

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Introduction: Frailty is an increasingly recognized concept in the context of multiple sclerosis (MS), and is regarded as a potential factor influencing clinical outcomes such as disability progression and disease activity. However, the concept of frailty in MS remains underexplored, and evidence regarding the use of standardized approaches to assess and capture frailty in this patient population is scarce.

Objective: The aim of this study was to evaluate the current evidence on frailty indices in MS populations, to investigate their validity and applicability, and to assess whether current evidence supports their integration into clinical practice.

Method: A systematic review was conducted following predefined eligibility criteria. Studies published between 2014 and 2024 that reported original data or systematic reviews on measures of frailty in patients with multiple sclerosis (pwMS) were included. Studies solely involving healthy individuals, patients with other diseases, or measures unrelated to frailty were excluded. Information sources included MEDLINE, EMBASE, and the Cochrane Library. The search strategy combined the terms "multiple sclerosis" and "frailty," filtered for English-language articles. Quality of evidence was assessed by 4 raters using the GRADE approach.

Results: Out of 817 screened studies, 10 met the inclusion criteria, comprising a total of 1941 pwMS. Three frailty indices were identified: Fried Frailty (FF), Frailty Index (FI), and Tilburg Frailty Indicator (TFI), with FI being the most studied (8

studies; 1653 patients across all studies). The FI showed significant associations with MS outcomes, including increased fall risk (IRR = 3.33; 95 % CI, 1.85–5.99; $p < 0.001$), lower likelihood of relapse (adjusted OR = 0.69; $p < 0.01$), and higher disability (EDSS; $\beta = 0.47$, $R^2 = 0.26$, $p < 0.001$). However, the quality of evidence was graded as low due to a high risk of bias (cross-sectional designs), lack of standardization (variations in indices, scoring thresholds, and data sources), imprecision (patient-reported data), and potential indirectness (scoring difference).

Both FF and TFI correlated with higher disability (FF: $p < 0.001$; TFI: $\beta = 0.57$; $R^2 = 0.35$, $p < 0.001$), and TFI was strongly associated with poorer quality of life ($p < 0.001$) and greater autonomy impairment ($p = 0.017$). However, their evidence was graded as very low due to a highly limited number of studies that did not allow for sufficient validation, risk of bias (cross-sectional study designs), and potential measurement inconsistencies (patient-reported data).

Feasibility of implementation in clinical routine varied. Although all indices are cost- and time-effective, the FF and FI require data not routinely available (e.g., grip strength, extensive questionnaires) and might require trained personnel, limiting their use in clinical practice. The TFI is more practical (self-obtained questionnaire) but constrained by insufficient MS-specific validation and potential reporting bias.

Conclusion: This review highlights the potential utility of frailty indices in MS while underscoring significant gaps in validation and subsequent applicability within this patient cohort. Current evidence suggests that these indices remain primarily aspirational tools, with their routine implementation in clinical practice being premature. Further studies are needed to validate and develop MS-specific frailty assessment tools.

P15: Anti-IgLON5 disease: A systematic review and meta-analysis – Clinical aspects

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Background: After its first description in 2014, anti-IgLON5 disease is now considered a complex and heterogeneous neurological disorder with sleep, bulbar, movement, and neuroimmunological as well as neurodegenerative aspects.

Aim: With a rapidly expanding spectrum of clinical presentations in anti-IgLON5 disease, the classification of clinical features becomes increasingly difficult. Therefore, the aim of this systematic review and meta-analysis was to entail the whole clinical spectrum as well as laboratory characteristics, therapeutic interventions, and reported outcomes of anti-IgLON5 disease.

Method: The electronic databases PubMed/MEDLINE, Web of Science, and Semantic Scholar were searched for case reports and case series on anti-IgLON5 disease. The search was last updated on July 31 2024. Only case reports or case series reporting on patients with positive IgLON5 antibodies in serum or cerebrospinal fluid (CSF) published in English were included for the systematic review. For data synthesis in the meta-analysis, only case series with $n \geq 10$ patients were considered.

Results: A total of 285 patients (169 patients from case series with $n \geq 10$ patients and 116 patients from single case reports and case series with $n < 10$ patients) were included in this study. A huge heterogeneity in the clinical presentation of patients with anti-IgLON5 disease was observed in our systematic review and meta-analysis, with sleep abnormalities (n = 218, 76.5 %), bulbar dysfunction (n = 175, 61.4 %) and movement disorders (n = 160, 56.1 %) being the most frequently reported features in this disorder. Considering movement disorders in anti-IgLON5 disease, a more detailed analysis of individual case reports showed that hyperkinetic disorders were reported most often (n = 48).

Conclusion: Due to the huge heterogeneity in its clinical presentation, the recognition and diagnosis of anti-IgLON5 disease represents a huge challenge in clinical routine. Based on this systematic review and meta-analysis, anti-IgLON5 disease should always be considered in patients presenting with sleep disorders and additional neurological symptoms that might resemble other diseases like progressive supranuclear palsy or amyotrophic lateral sclerosis but do not fulfill the respective diagnostic criteria or show additional features that are not typical for the presumed diagnosis.

P16: Real-life use of alemtuzumab, cladribine, dimethylfumarate, fingolimod, natalizumab, ocrelizumab, ofatumumab, ozanimod, ponesimod, siponimod, and teriflunomide: Benefit-risk data from the Austrian Multiple Sclerosis Treatment Registry

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Background: The efficacy of alemtuzumab, cladribine, dimethylfumarate (DMF), fingolimod, natalizumab, ocrelizumab, ofatumumab, ozanimod, ponesimod, siponimod, and teriflunomide in the treatment of multiple sclerosis (MS) has been proven in randomized trials. However, such trials do not necessarily reflect real-life situations faced in everyday practice.
Method: The baseline documentation within the Austrian MS Treatment Registry (AMSTR) includes duration of disease, relapses within the past 12 months, EDSS, MRI activity, and previous disease-modifying therapies. Entry of follow-up data (relapses, EDSS, adverse events) is required at 3-month intervals. In addition, changes in treatment are documented. The statistical values below indicate mean (range), unless otherwise indicated.
Results: As of December 20 2024, the registry comprised 37 patients started with alemtuzumab (65 % female), 177 patients with cladribine (71 % female), 2115 patients with DMF (70 % female), 1406 patients with fingolimod (69 % female), 1794 patients with natalizumab (71 % female), 383 patients with ocrelizumab (54 % female), 297 with ofatumumab (66 % female), 194 with ozanimod (64 % female), 79 with ponesimod (51 % female), 111 with siponimod (66 % female), and 623 patients with teriflunomide (64 % female). Altogether, 1414 patients switched within the AMSTR, most from natalizumab to fingolimod. At baseline, the mean age was 31.9 (18–50) years in the alemtuzumab, 35.7 (18–61) years in the cladribine, 36.8 (15–73) years in the DMF, 38.5 (13–72) years in the fingolimod, 34.8 (14–67) years in the natalizumab, 42.7 (18–76) years in the ocrelizumab, 35.2 (19–66) years in the ofatumumab, 38.8 (18–67) years in the ozanimod group, 36.8 (21–56) years in the ponesimod group, 52.7 (26–71) years in the siponimod group, and 43.2 (16–77) years in the teriflunomide group, with disease durations of 3.1 (0–15), 6.3 (0–39), 5.4 (0–56), 9.1 (0–44), 6.9 (0–40), 5.1 (0–36), 4.6 (0–32), 5.4 (0–36), 4.8 (0–25), 16.4 (0–39), and 8.1 (0–37) years respectively. The relapse rate in the year before start of respective drugs was 2.1 with alemtuzumab, 1.2 with cladribine, 1.0 with DMF, 1.4 with fingolimod, 2.1 with natalizumab, 0.7 with ocrelizumab, 1.1 with ofatumumab, 1.0 with ozanimod, 1.1 with ponesimod, 0.5 with siponimod and 0.7 with teriflunomide. For those treated for at least 1 year, the subsequent annualized relapse rates decreased to 0.2 (fingolimod, alemtuzumab), 0.1 (cladribine, DMF, natalizumab, ozanimod, ponesimod, and teriflunomide), and < 0.05 (ocrelizumab, ofatumumab and siponimod).
Conclusion: For more than 19 years, the AMSTR has proved valuable to measure quality of care and monitor treatment, providing neurologists with highly relevant information for clinical practice. Continuous optimization and extension of this registry represents a unanimous goal and necessity. Therefore, new treatment modules are currently being developed and data monitoring and communication will be further improved. The availability of an increasingly broad treatment armamentarium with its consequences for daily practice (e.g., monitoring long-term benefit/risk profiles of individual drugs and of their sequential use) emphasizes the need and the crucial importance of this registry for improved real-life management of MS patients in Austria.

P17: The role of age in choosing high-efficacy treatment for multiple sclerosis: An Austrian MS Database study

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Background: Treatment strategy for relapsing multiple sclerosis (RMS) is increasingly shifting toward first-line use of high-efficacy disease-modifying treatment (H-DMT). However, the efficacy of DMT appears to decline with increasing age, and the benefit of using H-DMT as first-line treatment at higher age currently remains unclear.
Objective: To investigate whether the superiority of H-DMT over moderate-efficacy DMT (M-DMT) as first-line treatment depends on age.
Method: Using the Austrian MS database, we included previously DMT-naive RMS patients aged ≥ 18 years, who i) initiated a DMT continuing it for ≥ 12 months; ii) had MRI at baseline; and iii) had clinical follow-up for ≥ 24 months (or occurrence of disease activity). The primary endpoint was the occurrence of relapse during follow-up. Age as well as DMT strategy (H-DMT vs. M-DMT) plus an interaction effect were employed as covariates in Cox regression analysis adjusting for sex, disease duration, disability assessed by the Expanded Disability Status Scale (EDSS) score at baseline, and number of relapses in the prior year, as well as number of hyperintense lesions on T2-weighted MRI (T2L) at baseline.
Results: A total of 215 RMS patients, median age of 41 (25th–75th percentiles: 32–53) years, 66 % female, were observed over a period of 42 (28–58) months. Eighty-one (38 %) patients had relapse. While patients' sex, disease duration, EDSS score at baseline, and number relapses in the prior year, as well as the number of T2L at baseline, had no statistically significant impact on the risk of relapse, increasing age was associated with decreased risk of relapse (hazard ratio (HR) 0.95, 95 % confidence interval (CI): 0.93–0.98, per year, $p < 0.001$). The use of H-DMT lowered the risk of relapse compared to M-DMT (hazard ratio (HR) 0.06, 95 %CI: 0.007–0.45, $p = 0.007$). However, in patients with H-DMT, the benefit of treatment was abrogated by increasing age (HR: 1.06, 95 % CI: 1.004–1.11, per year, $p = 0.032$).
Discussion: The benefit of H-DMT over M-DMT as first-line treatment decreased with increasing age and was lost in RMS patients above the age of approximately 50 years.

P18: Autoimmun-bedingte chronische Pachymeningitiden: Herausforderungen in Diagnose und Therapie anhand zweier Patientenfälle

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Hintergrund: Die Symptome und Ursachen der chronischen Pachymeningitis können je nach Lokalisation vielfältig sein und von Kopfschmerzen bis Hirnnervenparesen, Wesensveränderung, Anfällen und Fieber variieren. Neben einer ausführlichen körperlichen Untersuchung und genauen Anamnese sind breite und ggf. mehrfache Liquordiagnostik und Bildgebung der Neuroachse und des Thorax notwendig. In vielen Fällen ist eine Biopsie der Dura und der Meningen notwendig.
Fragestellung: Autoimmun-mediierte chronische Pachymeningitiden stellen diagnostisch und therapeutisch eine erhebliche Herausforderung dar. Histologische Befunde sind häufig unspezifisch. Inwiefern kann eine Kombination aus klinischen, bildgebenden und histopathologischen Befunden zur gesicherten Diagnose und gezielten Therapie führen?
Methode: Hier werden 2 Patientenfälle mit diagnostizierter chronischer Pachymeningitis vorgestellt.
Ergebnisse: Patient 1 war ein 49-jähriger Mann, der nach einer Facialisparese rechts im Rahmen einer Otitis media mit Osteomyelitis und konservativer Behandlung an der HNO-Abteilung mit zunehmender Dysphagie, Doppelbildern, Kopfschmerzen und mehrfachen Hirnnervenparesen vorstellig wurde. Im MRT war ein entzündlicher Prozess an der Schädelbasis ▶

nachweisbar. Die Liquordiagnostik zeigte 75 Zellen/µl, die MPO-Antikörper waren gering erhöht. Eine Durabiopsie ergab einen unspezifischen entzündlichen Prozess. Aufgrund einer positiven Streptokokken-species-PCR im Biopsiematerial erfolgte ein Ausschleichen der bereits begonnenen Cortisontherapie und eine antibiotische Therapie bei V. a. fortgeleiteten erregerbedingten Prozess. Bei klinischer Verschlechterung unter Antibiose erfolgten eine neuerliche Abklärung und ein Wiederbeginn der Cortisontherapie mit gutem Ansprechen. Somit wurde die Diagnose einer MPO-AK-assoziierten chronischen Pachymeningitis gestellt und eine immunsuppressive Therapie mit 12 Zyklen Cyclophophamid gefolgt von Rituximab über 3 Jahre etabliert. Der Patient stabilisierte sich klinisch.

Patientin 2, eine 38-jährige Frau mit bekannter seropositiver rheumatoider Arthritis unter immunsuppressiver Therapie, präsentierte sich mit fokalen motorischen Anfällen und einer Meningoenzephalitis im Interhemisphärenspalt frontal im MRT. Im Liquor zeigte sich eine Pleozytose mit 69 Zellen/µl. In der Abklärung zeigte sich eine zystische Raumforderung in der Niere, die parasitologisch untersucht wurde. Die Patientin verschlechterte sich zunehmend und entwickelte einen Status epilepticus. Aufgrund der klinischen Verschlechterung unter breiter antibiotischer Therapie wurde zusätzlich eine Kortikosteroidtherapie begonnen und eine Biopsie mit Befundung am Klinischen Institut für Neurologie, AKH Wien, organisiert. Diese ergab einen granulomatös-nekrotisierenden Prozess mit Plasmazellen, tlw. IgG4 positiv, vereinbar mit einer Meningitis assoziiert mit rheumatoider Arthritis. Der MRT-Befund besserte sich unter Cortison. Es wurde eine Therapie mit Rituximab über 4 Jahre etabliert, nach der die Patientin bezüglich der Meningitis stabil blieb.

Patient 1 zeigte, dass neben einer Durabiopsie, die manchmal unspezifisch ist, auch die Klinik in Zusammenschau mit auch nur gering erhöhten Antikörpern wegweisend sein kann.

Patientin 2 erhielt eine differenziertere neuropathologische Befundung, die diagnostisch hilfreich war. Der frühzeitige Therapiebeginn führte zu einer guten klinischen Remission. Eine CD20-AK-Therapie mit Rituximab wurde gewählt, da die Patientin vorbestehend immunsupprimiert war und insbesondere bei fehlendem Ansprechen auf orale Immunsuppressiva eine B-Zell-depletierende Therapie aufgrund des Vorliegens von zahlreichen Plasmazellen in der Histologie empfohlen wird.

Zusammenfassung: Autoimmunbedingte Pachymeningitiden stellen eine diagnostische Herausforderung dar, da histologisch oft unspezifische Befunde vorliegen und sie schwer von anderen entzündlichen Prozessen zu unterscheiden sein können. Eine frühzeitige und differenzierte Diagnosestellung in Zusammenschau mit der Klinik ist entscheidend. Neben Kortikosteroiden ist meist eine immunsuppressive Therapie notwendig.

P19: Diagnostic accuracy of inter-eye difference of ganglion cell layer alone in identifying optic neuritis in multiple sclerosis

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Introduction: The 2024 McDonald criteria for diagnosing multiple sclerosis (MS) include optic nerve involvement as a fifth region for establishing dissemination in space. Optic neuritis (ON) can be detected through optical coherence tomography (OCT) using an inter-eye absolute or percentage difference (IEAD, IEPD) in ganglion cell-inner plexiform layer (GCIPL) thickness.

Objective: To compare the diagnostic accuracy of GCIPL IEAD/IEPD with GCL and IPL IEAD/IEPD alone for identifying a history of ON.

Method: This cross-sectional retrospective study included people with MS (pwMS) who underwent an OCT scan. Diagnostic accuracy was assessed using ROC analysis.

Results: A total of 241 pwMS (mean age 34.7 years [SD 9.7], 70.1 % female) were included. GCL IEAD (AUC 0.88, cut-off ≥ 0.04mm3 or ≥ 1.4µm, 80.0 % sensitivity, 86.5 % specificity) and IEPD (AUC 0.89, cut-off ≥ 4 %, 79.0 % sensitivity, 87.2 % specificity) demonstrated excellent diagnostic accuracy for unilateral ON, showing non-inferiority to the established GCIPL IEAD/IEPD. An improvement in diagnostic performance of both models was observed in a subanalysis of pwMS with subclinical ON (AUC 0.95, sensitivity 93.8 %, specificity 87.2 %).

Conclusion: GCL IEAD and IEPD provide strong diagnostic accuracy for identifying unilateral ON and can be effectively used as an alternative to GCIPL IEAD/IEPD to facilitate implementation in clinical routine.

P20: Serum neurofilament light chain and glial fibrillary acidic protein levels are associated with inner retinal layer thinning in multiple sclerosis

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Background and Objective: Serum neurofilament light chain (sNfL) and glial fibrillary acidic protein (GFAP) are emerging biomarkers of axonal damage and astrocytic activation, both likely paramount in multiple sclerosis (MS) associated neurodegeneration. However, the value of sNfL and GFAP in predicting inner retinal layer thinning, a reliable surrogate of neuroaxonal damage, remains underexplored. We aimed to evaluate the association between sNfL and GFAP levels and inner retinal layer thinning.

Method: This prospective observational study included patients with relapsing MS newly initiated on a disease-modifying therapy (DMT). OCT scans were conducted 3–6 months after DMT initiation (rebaseline) and at 12-month intervals, measuring peripapillary retinal nerve fiber layer (pRNFL) and ganglion cell-inner plexiform layer (GCIPL) thickness. Serum sNfL and GFAP levels were measured at baseline and after 6 months using single molecule array (Simoa) tech- ▶

nology, with z-scores adjusted for age, BMI, and—for GFAP—sex. Linear regression models were employed to calculate the annualized loss of pRNFL (aLpRNFL) and GCIPL (aLGCIPL), and to determine their associations with sNfL and GFAP.

Results: A total of 116 patients (mean age 34.5 years [SD 8.6]), 73.3 % female, median disease duration 2.8 years [IQR 0.3–7.1], median EDSS 1.5 [range 0–5.5]) were included. Both pRNFL (b= -0.34; 95 % CI -0.67, -0.02; p = 0.04) and GCIPL thicknesses (b= -0.39; 95 % CI -0.65, -0.12; p = 0.004) were associated with GFAP—but not sNfL—z-scores at baseline. GFAP z-scores at M6 showed the strongest association with aLpRNFL (b= -0.24; 95 % CI -0.27, -0.21, p < 0.001) and aLGCIPL (b= -0.15; 95 % CI -0.18, -0.12, p < 0.001). Moreover, patients with low sNfL but high GFAP levels at M6 showed the most pronounced inner retinal layer thinning (aLpRNFL: -0.8 %/year [1.1], aLGCIPL: -0.8 %/year [0.9]; both p < 0.001).

Conclusion: High GFAP levels—more than sNfL levels—are associated with inner retinal layer thinning in RMS, underscoring their value as biomarkers of disease progression.

P21: Temporal dynamics of serum neurofilament light chain in multiple sclerosis: A retrospective study in a clinical routine setting

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Background: Serum neurofilament light chain (sNfL), a biomarker for neuroaxonal injury, has been strongly associated with disease activity in multiple sclerosis (MS). Elevated sNfL levels have been linked to evidence of disease activity (EDA) compared to clinically and radiologically stable patients. However, the temporal dynamics of sNfL in relation to disease relapses and its clinical implications in routine settings remain poorly understood.

Objective: This study aims to evaluate the temporal changes in sNfL levels in relation to MS relapsing activity and to elucidate their potential utility in clinical assessments.

Method: Retrospective longitudinal data from 162 MS patients (mean age 32.5 ± 7.8 years, median [IQR] disease duration 2.1 [1–7.1], 64.2 % female) were analyzed, with a median of 7 (IQR 6–9) serum samples per patient collected over a median follow-up time of 10.4 (7.8–13.8) years. sNfL levels were quantified using the Simoa HD-X analyzer, and results were adjusted for age and body mass index using z-scores. Radiological activity was assessed through gadolinium-enhanced lesions detected using 3T MRI scans. EDA has been defined as the occurrence of any of the following within6 months of sampling: clinical relapses, confirmed disability worsening as assessed by Expanded Disability Status Scale (EDSS) scores, or radiological activity.

Results: sNfL z-scores were significantly elevated in patients with future EDA occurring within 1 year of sample collection, but only when samples were taken during remission (p < 0.001). Additionally, sNfL levels did not predict EDA beyond this one-year window. The temporal analysis around clinical relapses showed an increase in sNfL z-scores at relapse onset (p < 0.001), with these elevated levels persisting for up to 9 months post-relapse.

Conclusion: These findings highlight the importance of monitoring sNfL as a dynamic biomarker for disease activity in MS. Accurate knowledge of the temporal dynamics of sNfL is essential for correct interpretation of sNfL in routine clinical practice and for identifying patients at risk of future disease activity. This approach could enhance clinical decision making and potentially improve routine MS care.

P22: Real-world use of disease-modifying therapies in Austria: Safety data from the Austrian multiple sclerosis Treatment Registry (AMSTR)

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Background: In the therapeutic landscape of multiple sclerosis (MS), more than a dozen disease-modifying therapies (DMT) are currently available. In Austria, only certified MS centers are authorized to prescribe DMT, and these centers are required to enter patient data into the Austrian MS Therapy Registry (AMSTR), which was established in 2006 to gain real-word evidence on the effectiveness and safety profile of these DMT. Here, we report the safety profiles of different DMT documented in the AMSTR.

Method: The data obtained from the AMSTR, spanning August 2006 to December 2020 and collected at least biannually, included adverse events (AE) among others. AE were classified by system organ classes according to the Medical Dictionary for Regulatory Activities.

Results: This analysis included 5134 patients (68.2 % female, median age 37 years [29–45]), with the majority receiving fingolimod (1969/5134, 38.4 %), followed by natalizumab (1760/5134, 34.3 %), and dimethyl fumarate (1552/5134, 30.2 %). Among these patients, 1317/5134 (25.7 %) reported at least 1 AE, with gastrointestinal symptoms (390/1317, 29.6 %) and infections (354/1317, 26.9 %) being the most common. For alemtuzumab, the most frequent AE were immune-related (38/88, 43.2 %) including thyroid disorders (21/88, 23.9 %). For cladribine, natalizumab, and ocrelizumab, infections were the most frequent (6/154, 3.9 %; 112/1760, 6.4 %; 9/260, 3.5 %). Gastrointestinal AE were most frequent with dimethyl fumarate and teriflunomide (250/1552, 16.1 %, 62/577, 10.7 %), whereas blood system-related AE were most common with fingolimod (195/1969, 9.9 %). Regarding neoplasms, all DMT showed a frequency of < 1 %. Overall, AE were most prevalent in alemtuzumab-treated patients (56/88, 63.6 %), which also showed the highest frequency of severe AE (SAE 4/88, 4.5 %).

Conclusion: The safety data align with previously published data on respective DMT and do not reveal any new safety issues, particularly regarding neoplasms and infections. Use of alemtuzumab is limited by the European Medicines Agency to adult patients with rapidly evolving severe RRMS or inadequate response to high-efficacy DMT, and requires rigorous safety monitoring.

P23: The value of oligoclonal bands in multiple sclerosis compared to other neurological disorders: A retrospective data analysis from an Austrian tertiary center

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Background: Cerebrospinal fluid (CSF) analysis represents a key pillar for diagnosing multiple sclerosis (MS). Alongside differential diagnostic aspects, in particular the presence of CSF-specific oligoclonal bands (OCB) is examined. Hence, the aim of this work is to investigate the value of OCB in MS compared to other neurological disorders with special emphasis on their value for differential diagnostic considerations.

Method: Patients who presented to the Department of Neurology of the Medical University of Vienna between January 1 2004 and December 31 2020 due to neurological complaints and who underwent lumbar puncture (LP) for diagnostic purposes were included in this retrospective analysis. Exclusion criteria were incomplete laboratory data and/or a missing final diagnosis. ▶

Results: Of 5744 patients who underwent LP, 5225 were included in the analysis. Of these, 745 were MS patients in whom CSF-specific OCB showed a sensitivity of 92.8 % and a specificity of 90.4 %. Further, the positive predictive value (PPV) was merely 61.5 %, whereas the negative predictive value (NPV) yielded 98.7 %. In comparison, most other neurological disease groups showed low sensitivity rates. Of note, central nervous system (CNS) borreliosis showed a relatively high OCB positive rate of 70 %, followed by CNS human immunodeficiency virus infections with 62.5 %.
Conclusion: CSF-specific OCB show a high sensitivity and specificity and a very high NPV for the diagnosis of MS. However, the PPV was significantly lower in this cohort. These findings need to be carefully considered when making a diagnosis of MS, especially in the context of differential diagnostic work up.

P24: Cognitive dysfunction during acute inflammation in multiple sclerosis: Disease activity related or mere sickness behavior?

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Background: Despite the recognition of the clinical importance of cognitive impairment (CI) even in early multiple sclerosis (MS), little is known about the dynamics of CI during acute MS relapse vs. a stable disease phase.
Objective: To assess CI in MS patients during acute relapse and its development over a 6-month follow-up.
Method: Adult people with MS (pwMS) with an acute relapse were recruited in the outpatient clinic of the department of neurology at Medical University of Vienna. Within the scope of this, the following assessments were conducted before administration of high-dose methylprednisolone (HDMP): Expanded Disability Status Scale (EDSS), neuropsychological testing including the Symbol Digit Modalities Test (SDMT), patient-reported outcomes (PROs) including self-assessment of cognition (MSNQ), fatigue (FSMC), depression and anxiety (HADS). Furthermore, magnetic resonance imaging (MRI), optic coherence tomography, and evoked potentials, as well as serum biomarker including neurofilament light chain (NfL) and glial fibrillary acidic protein were examined.
Results: Fifty-five pwMS with acute relapse were included, with 74 % (n = 40) being female and 30 % (n = 16) with an initial manifestation of MS. Median age was 34 years (29–41), disease duration was 3 years (0–12), and median EDSS was 3.0 (2.5–3.5). According to the SDMT, 17 % were cognitively impaired (z-score < -1.5) and 39 % had a borderline result (z-score > -1,5 - < -0.5). PwMS with CI (SDMT z-score < -1.5) had a significantly (7.76cm^3 [5.85cm^3–25.61cm^3] vs. 2.29cm^3 [1.71cm^3–6.46cm^3], p = 0.004, d = 1.5) higher T2-lesion volume on MRI at baseline. At 6 months follow-up (n = 51), 55 % showed an improvement in the SDMT of 4 or more points. Improvers displayed a significantly lower score in the PRO for self-assessment of cognition (p = 0.025, d = 0.75) after 6 months in comparison to baseline and non-improvers and no significant differences regarding the interactors depression, anxiety, and fatigue. Moreover, improvers, compared to non-improvers, showed a significant reduction in T2-lesion volume on MRI (p = 0.021, d = -0.43)

and a numerically higher NFL z-score at baseline (p = 0.056, d = 0.68) as well as a shorter disease duration (p = 0.055, d = -0.55). In addition, in the group of SDMT improvers a higher proportion of pwMS was without DMT at relapse (64 % vs. 43.5 %) and was more often treated with a highly effective therapy (50 % vs. 26.1 %) at 6-month follow-up. **Conclusion:** CI during relapse is a frequent—although at least partially reversible—phenomenon. Improvement of CI based on SDMT was not driven by confounders like depression, anxiety ,or fatigue and was in line with self-assessment of cognition. Clinical and paraclinical findings suggest that CI in MS does not only reflect disease progression but is also a relapse-related phenomenon, prone to potential remission thereafter. Long-term follow-up of this cohort is already ongoing in order to explore this in depth.

P25: Do people with multiple sclerosis age differently? Reference values for NK-cell aging and study outline

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Background: Chronological age is the most consistent factor influencing the observed disease course of people with multiple sclerosis (pwMS). Furthermore, the efficacy and safety of disease-modifying treatments (DMT) vary considerably in older individuals. Insights into the biological age of pwMS are not only essential for advancing our understanding of MS course and pathophysiology but may also hold meaningful practical implications for individualized treatment decisions. The expression of the marker CD57 on CD56dim natural killer (NK) cells and its proportion of the whole NK population is a well-established indicator for matured NK cells and may therefore serve as a surrogate marker for biological age.

Aim: To investigate differences in biological aging and immunosenescence between pwMS and healthy controls (HCs) and to identify potential covariates associated with an accelerated biological aging process in pwMS.

Method: Peripheral blood mononuclear cells (PBMCs) from healthy blood donors in Switzerland, Austria, and Germany were analyzed using flow cytometry, gating for CD3, CD56, and CD57. Age-adjusted z-scores for proportions of CD57+CD56dim and CD56bright NK cells were estimated using a generalized additive model for location, scale, and shape (GAMLSS). In this ongoing study, PBMCs from 100 pwMS will be collected to enable a direct comparison of immunobiological NK-cell aging. Furthermore, potential covariates of accelerated cell aging, such as disease duration, treatment duration, and DMT type, will be evaluated in 80 pwMS across 16 distinct subgroups.

Results: Data from 10437 HCs aged 18–70 years (mean age 45.9 years [SD 13.3], 33.6 % female) were analyzed. The mean proportion of CD57+CD56dim cells was 38.4 % (SD 15.4) and the mean proportion of CD56bright cells was 9.1 % (SD 13.3). There was a fourfold increase in CD57+CD56dim NK cells when comparing individuals younger than 21 with those older than 64 years, whereas the proportion of immunoregulatory CD56bright cells decreased by 23.1 %. On average, the proportion of CD57+CD56dim NK cells increased by 3.9 % per year. Adding sex as a covariate did not show a statistically significant association with either proportion and did not improve the model fit, as indicated by the Akaike Information Criterion and likelihood ratio tests.

Conclusion: CD57+CD56dim NK cells are a robust marker for NK cell aging and may serve as a reliable surrogate for biological aging regardless of sex. Based on these findings, age-adjusted z-scores for both cell proportions can be predicted for pwMS to assess whether biological and chronological age differ, and which covariates account for most of the variation. Once data collection for pwMS is complete, these exploratory results will offer initial insights into the aging process and immunosenescence, forming the foundation for future studies.

P26: Arteriosklerotische Veränderungen bei älteren Patient:innen mit Multipler Sklerose

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Hintergrund: Die Multiple Sklerose (MS) ist eine der häufigsten neurologischen Erkrankungen junger Erwachsener und die häufigste neurologische Autoimmunerkrankung. Aufgrund der immer besser werdenden Diagnostik, Therapie und Prognose gibt es immer mehr ältere Patient:innen mit MS (PwMS). Die Lebenserwartung ist nur um wenige Jahre verkürzt, weshalb sich die Frage stellt, wie es um das vaskuläre Langzeitrisiko von Betroffenen steht. Die „MSVasc"-Studie beschäftigt sich mit älteren PwMS zwischen 50 und 60 Jahren und zielt darauf ab, vaskuläre Risikoparameter zu erfassen. Einen wichtigen Teil der Untersuchungen während der Studienvisiten stellte die Neurosonografie dar, mit der sich diese Auswertung befasste.
Fragestellung: Ziel dieser Auswertung war es, die sonografischen Bilder der Baseline-Visiten auszuwerten und den Zustand der hirnzuführenden extrakraniellen Arterien zu beurteilen. Der Fokus lag auf arteriosklerotischen Veränderungen der Gefäße, wie Zunahme der Intima-Media-Dicke (IMD) oder Formation von Plaques. Mithilfe weiterer Variablen wie Alter, Geschlecht, BMI oder Raucherstatus wurde versucht, eine Abschätzung des vaskulären Risikos zu tätigen.
Methoden: Diese Auswertung ist Teil einer prospektiven, longitudinalen Kohortenstudie, die PwMS zwischen dem 50. und 60. Lebensalter inkludierte. In dieser Auswertung wurden die sonografischen Daten von 97 Patient*innen analysiert. Das Ziel dieser Auswertung war, deskriptive Statistiken zu den sonografischen Daten zu präsentieren, diese mit einigen klinischen und demografischen Variablen zu korrelieren und mit Referenzwerten aus der Literatur zu vergleichen.
Ergebnisse: Die Studie inkludierte 97 Patient*innen (25,8 % männlich, 74,2 % weiblich). Insgesamt wurden bei 40 % Plaques gefunden. 48 % der Männer und 37,5 % der Frauen hatten Plaques. 80 % der Männer und 63,9 % der Frauen hatten eine erweiterte IMT. Diese Werte sind allesamt über den erwarteten Werten in der Normalpopulation, was auf ein höheres vaskuläres Risiko in der Studienpopulation hindeuten könnte. Als statistisch signifikanter Risikofaktor für das Auftreten von Plaques konnten die arterielle Hypertonie ($p = 0{,}023$, $V = 0{,}231$) sowie das Alter ($p = 0{,}010$, $r = 0{,}262$) identifiziert werden. Ebenso konnte eine schwache Korrelation zwischen dem Plaquedurchmesser und dem Rauchverhalten ($p = 0{,}042$, $r^2 = 0{,}110$), der sonografisch erfassten Plaquenanzahl ($p = 0{,}045$, $r^2 = 0{,}107$) sowie dem Rauchverhalten in Kombination mit dem EDSS ($p = 0{,}007$, R korr. $= 0{,}203$) und der MS-Form ($p = 0{,}012$, R korr. $= 0{,}179$) gezeigt werden.
Zusammenfassung: Die Studienpopulation zeigte eine unerwartet hohe Prävalenz von vaskulären Risikoparametern wie Plaqueformation, Rauchen oder erweiterte IMT. Dies deutet auf ein erhöhtes vaskuläres Risiko für die Studienpopulation hin und zeigt die Wichtigkeit von regelmäßigen Kontrollen der vaskulären Parameter bei PwMS. Ebenso konnte eine schwache Korrelation zwischen MS-spezifischen Parametern und der klinischen Signifikanz von Plaques gezeigt werden, was bedeuten könnte, dass die MS neben dem zentralen Nervensystem auch das vaskuläre System beeinflussen und vaskuläre Dysfunktion fördern könnte.

P27: Hat die kognitive Fatigue bei MS-Patient*innen im Akutschub einen direkten Einfluss auf die verbale Enkodierungs- bzw. Konsolidierungsleistung?

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Hintergrund: Fatigue stellt eine der häufigsten Begleiterscheinungen von Menschen mit Multipler Sklerose (PwMS) dar und wird subjektiv als überaus belastend wahrgenommen. Verschiedene Domänen der kognitiven Leistungen können durch den Mediator Fatigue erheblich negativ beeinflusst werden.
Fragestellung: Welche Bedeutung hat

die „Trait"-Komponente der kognitiven Fatigue bei PwMS im Zusammenhang mit kognitiver Fatigue und Enkodierungs- bzw. Konsolidierungsleistung in der Akutphase der MS?

Methode: 52 PwMS mit überwiegend schubförmig remittierendem Verlauf (RRMS) wurden bei Vorliegen eines akuten Schubes vor Beginn der kostikosteroiden Akutbehandlung auf kognitive Fatigue und kognitive Leistungsdefizite untersucht. Dafür wurden die Fatigue-Skala für Motorik und Kognition (FSMC), die Testbatterie zur Aufmerksamkeitsprüfung (TAP), der Symbol Digit Modalities Test (SDMT), der Verbale Lern- und Merkfähigkeitstest (VLMT) sowie „Zahlen nachsprechen rückwärts" der Wechsler Adult Intelligence Scale (WAIS-IV) verwendet. Es wurden PwMS ohne bzw. mit leicht ausgeprägter kognitiver Fatigue von solchen mit mittelgradig oder schwer ausgeprägter kognitiver Fatigue in ihrer Gedächtnisleistung unterschieden. Des Weiteren wurde untersucht, ob der Zusammenhang zwischen kognitiver Fatigue und Gedächtnisbeeinträchtigungen über die beiden Aufmerksamkeitskomponenten Alertness und kognitive Verarbeitungsgeschwindigkeit mediiert wird.

Ergebnisse: Bei der Überprüfung eines indirekten Effekts von kognitiver Fatigue und Gedächtnisbeeinträchtigungen über Alertness und kognitive Verarbeitungsgeschwindigkeit zeigte sich lediglich ein indirekter Effekt für den Zusammenhang von kognitiver Fatigue und der Lernleistung des verbalen Gedächtnisses, der über die kognitive Verarbeitungsgeschwindigkeit vermittelt wird.

Schlussfolgerung: Der signifikante Mediationseffekt deutet darauf hin, dass eine stärkere Berücksichtigung von kognitiver Fatigue und kognitiver Verarbeitungsgeschwindigkeit bei zukünftigen Behandlungsinterventionen von PwMS erfolgversprechend sein könnte.

28: HLA-dependency and possible clinical relevance of intrathecally synthesized anti-IgLON5 IgG4 in anti-IgLON5 disease

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Background: Anti-IgLON5 disease is a rare chronic autoimmune disorder characterized by IgLON5 autoantibodies predominantly of the IgG4 subclass. Distinct pathogenic effects were described for anti-IgLON5 IgG1 and IgG4, however with uncertain clinical relevance. Prognostic biomarkers and treatment guidelines are lacking and urgently needed.

Aim: We aimed to evaluate the role of anti-IgLON5 IgG4 and IgG1 levels in serum and CSF in correlation to clinical parameters.

Research questions: questions: Are IgLON5 IgG1 or IgG4 levels in serum or CSF potential biomarkers? Which treatment(s) are associated with clinical improvement and/or reduction of autoantibody levels? Are autoantibody levels associated with presence of the risk alleles HLA-DRB1*10:01 and DQB1*05:01?

Method: IgLON5-specific IgG1-4 levels were measured in 46 serum and 20 cerebrospinal fluid (CSF) samples from 13 HLA-subtyped anti-IgLON5 di- ▶

sease patients (6F, 7M) using flow cytometry. Intervals between 2 consecutive serum or CSF samplings (31 and 10 intervals, respectively) were categorized with regard to the immunomodulatory treatment active at the end of the interval, changes of anti-IgLON5 IgG1 and IgG4 levels, and disease severity. Intrathecal anti-IgLON5 IgG4 synthesis (IS) was assessed using a quantitative method.

Results: The median age at onset was 66 years (range 54–75), disease duration 10 years (range 15–156 months), and follow-up 25 months (range 0–83). IgLON5-specific IgG4 predominance was observed in 38/46 (83 %) serum and 11/20 (55 %) CSF samples. Anti-IgLON5 IgG4 levels prior clinical improvement in CSF but not serum were significantly lower than in those prior stable/progressive disease. Compared to IgLON5 IgG4 levels in serum, CSF levels in HLA-DRB1*10:01 carriers were significantly higher than in non-carriers. Indeed, IgLON5-specific IgG4 IS was demonstrated in 4/5 HLA-DRB1*10:01 carriers and in 1 non-carrier. Immunotherapy was associated with decreased anti-IgGLON5 IgG serum levels. In CSF, lower anti-IgLON5 IgG was associated with immunosuppressive treatments used in combination, i.e., corticosteroids and/or azathioprine plus intravenous immunoglobulins or rituximab.

Conclusion: Our findings might indicate that CSF IgLON5-specific IgG4 is frequently produced intrathecally, especially in HLA-DRB1*10:01 carriers. Intrathecally produced IgG4 may be clinically relevant. While many immunotherapies reduce serum IgLON5 IgG levels, more intense immunotherapy induces clinical improvement and may be able to target intrathecally produced anti-IgLON5 IgG. Further studies need to confirm whether anti-IgLON5 IgG4 IS is a suitable prognostic and predictive biomarker in anti-IgLON5 disease.

P29: Osteoporosis in multiple sclerosis: Validation of a new risk score

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Background: Osteoporosis is a highly relevant—but often underestimated and underdiagnosed—comorbidity in people with multiple sclerosis (pwMS) which negatively impacts morbidity. Although the prevalence is higher in pwMS compared to the general population, specific screening tools in the MS population are lacking. A newly developed risk score offers a simple way to calculate individual osteoporosis risk and guide screening recommendations.

Objective: To validate a previously developed risk score for its accuracy and clinical applicability in predicting the risk of osteoporosis in pwMS.

Method: In this prospective study, densitometry (hip and lumbar spine) was performed in pwMS who were seen at the MS clinic of the Department of Neurology of the Medical University of Innsbruck. The relevant risk factors (age, menopausal status, body mass index, smoking, disability assessed by the Expanded Disability Status Scale) were collected. Agreement between the risk score's prediction of osteoporosis and the actual detection of osteoporosis was compared.

Results: A total of 80 pwMS, at the mean age of 42 years and with a female predominance of 68 %, were included. A priori, the risk score identified 10 pwMS (12.5 %) as having an increased risk of osteoporosis (defined as a probability > 60 %). Of those, 3 pwMS indeed had osteoporosis as detected by densitometry (positive predictive value 30 %). Of the remaining 70 pwMS predicted as having no or low risk of osteoporosis, 68 showed normal bone mineral density by densitometry (negative predictive value 97 %).

Conclusion: The proposed score qualifies as a simple tool to identify pwMS with higher risk of osteoporosis, and to select those who should be prompted to densitometry. This personalized treatment approach should allow early treatment of osteoporosis and thus prevent fractures and morbidity in pwMS.

P30: Impact of renal function impairment on kappa free light chain index

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Introduction: Kappa free light chains in the cerebrospinal fluid (CSF) are an emerging diagnostic and prognostic biomarker in multiple sclerosis (MS). Impaired renal function leads to altered serum κ-free light chain (FLC) and albumin concentrations. Whether renal function has to be considered for the interpretation of κ-FLC in the CSF is unclear.
Objective: To investigate whether renal function impacts CSF κ-FLC concentration and/or κ-FLC index.
Method: Patients with non-inflammatory neurological diseases (NIND) with CSF white blood cells < 5/µl and negative oligoclonal banding were included. Glomerular filtration rate (GFR) was determined by the CKD-EPI equation. κ-FLC index was calculated as (CSF κ-FLC/serum κ-FLC)/(CSF albumin/serum albumin). Univariate correlation analyses were performed by Spearman correlation coefficient. Structural equation modeling (SEM) was used to evaluate the direct influence of GFR on serum κ-FLC concentration and CSF/serum albumin ratio (Qalb), and via these two variables the indirect influence on CSF κ-FLC concentration.
Results: A total of 129 NIND patients, with a median age of 65 years and 42 % female, were included in the study. κ-FLC index ranged from 0.57 to 3.56 and GFR ranged from 17 to 128 ml/min/1.73m^2. While a correlation of GFR with CSF κ-FLC concentration was observed ($r = -0.52$, $p < 0.001$), there was no statistically significant correlation with κ-FLC index ($r = 0.14$, $p = 0.113$). SEM revealed a causal chain: Higher age was associated with lower GFR ($\beta = -0.53$), which in turn led to higher serum κ-FLC concentration ($\beta = -0.45$) and higher Qalb ($\beta = -0.17$). CSF κ-FLC concentration increased with serum κ-FLC concentration ($\beta = 0.75$) as well as Qalb ($\beta = 0.39$), indicating that GFR did not directly influence CSF κ-FLC concentration (RMSEA = 0.043).
Conclusion: CSF κ-FLC concentration is not directly affected by renal function impairment. The κ-FLC index compensates for renal function effects by factoring in serum κ-FLC concentration and Qalb. Thus, ν-FLC index can be interpreted without considering renal function.

P31: Long-term stability of kappa free light chain index

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Background: Kappa free light chains (κ-FLC) in the cerebrospinal fluid (CSF) are a novel biomarker to assess intrathecal immunoglobulin synthesis. The impact of frozen storage duration on CSF κ-FLC is not known.
Objective: To investigate whether frozen storage duration influences CSF κ-FLC concentration and κ-FLC index.
Method: CSF and serum samples of patients with multiple sclerosis (n = 70) and Lyme disease (n = 12) collected for routine diagnostic purposes were stored at -20°C. κ-FLC and albumin concentrations were measured at 2 different timepoints, i.e., before (M1) and after (M2) storage. Agreement between measurements was analyzed by Passing-Bablok regression, and concordance between sample positivity by kappa statistics.
Results: A total of 82 patients with a median age of 33 years and a female predominance of 61 % were included. Median time between the measurements M1 and M2 was 2.3 years (75th percentile 4.3 years, maximum 7.4 years). Absolute concentrations of albumin and κ-FLC in CSF and serum did not show a meaningful change over time, with a percentage change of less than 2 % per year. Hence, κ-FLC index was also not statistically significantly changed by frozen storage duration ($\beta = -0.012$, per year, $p = 0.327$) showing comparable values at M1 (median 37, 25th–75th percentile 23–89) and M2 (35, 20–77). Cohen's kappa of the χ-FLC index between M1 and M2 was 0.85.
Conclusion: Frozen storage has no relevant impact on CSF κ-FLC concentration and κ-FLC index.

P32: Reduced muscle mass and risk of sarcopenia in multiple sclerosis

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Background: In recent years, there has been growing interest in sarcopenia and its connection to nervous system disorders. Multiple sclerosis (MS) related data on sarcopenia are limited. Various measurement methods are available, and temporal muscle thinning (TMT) has emerged as a reliable screening marker.
Objective: To investigate the risk of sarcopenia in people with MS (pwMS) and its association with disease-specific factors such as disability.
Method: In this prospective study, body mass index (BMI), abdominal circumference, body fat, and skeletal muscle mass measured by bio-electrical impedance analysis (Inbody 770) and possible risk factors, i.e., age, smoking, and disability assessed by the Expanded Disability Status Scale (EDSS), were collected. In addition, bone mineral density and osteoporosis was assessed by densitometry. Reduced muscle mass was calculated by Appendicular Skeletal Muscle Mass Index (ASMMI) and the risk of sarcopenia was screened via TMT from routine MRI.
Results: A total of 54 pwMS at the mean age of 45.9 years with a female predominance of 63 % and a median EDSS of 2.0 were included. The ASMMI was reduced in 24 (44.5 %) patients. Increased sarcopenia risk as per TMT was detected in 7 (13 %) pwMS. All pwMS with a reduced TMT also had a reduced ASMMI. Whereas no disease-specific factors were associated with an increased risk of sarcopenia, a lower BMI was identified as a risk factor (27.47kg/m^2 vs. 21.53kg/m^2; $p < 0.001$). In addition, in patients with a reduced muscle mass diagnosis, osteoporosis was significantly more frequent (n = 2, 6.7 % vs. n = 8, 33.3 %; p = 0.016) and bone mineral density was reduced (lumbar spine 1.11g/cm^2 vs. 1.00g/cm^2, p = 0.015; total femur 0.98g/cm^2 vs. 0.88g/cm^2, p = 0.004).

Conclusion: TMT is a quick and simple method to assess the risk of sarcopenia in MS. In this study, 13 % of pwMS had a positive screening for sarcopenia. In addition, nearly half of the pwMS had a reduced muscle mass. This was associated with a significantly higher osteoporosis risk, and these patients should be advised regarding dietary intake, exercise, and osteoporosis screening.

P33: Choroid plexus volume and serum neurofilament light levels in relation to brain atrophy and lesion load in multiple sclerosis

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Background: There is growing evidence that the choroid plexus, responsible for cerebrospinal fluid (CSF) production and the blood-CSF barrier, plays a significant role in inflammatory processes affecting the central nervous system (CNS). Research indicates that choroid plexus volume (CPV) is increased in patients with multiple sclerosis (pwMS) and is linked to clinical and MRI-based indicators of disease progression. Consequently, CPV is being considered as an imaging biomarker for disease monitoring. However, the extent to which CPV, alongside serum neurofilament light (sNfL), an established blood-based marker for neuroaxonal damage, predicts lesion load and brain atrophy in pwMS remains unclear.
Aim: This study aimed to determine how well CPV and sNfL indicate lesion burden and brain volume changes over a median follow-up of 5.3 years (IQR: 4.6–5.5).
Method: Ninety-six pwMS (17 with clinically isolated syndrome, 70 with relapsing-remitting MS, and 9 with progressive MS) and 49 age- and sex-matched healthy controls participated. Participants underwent 3T MRI to evaluate normalized brain volume and lesion load using FreeSurfer and SIENA. sNfL was measured with a Simoa HD-X analyzer. Longitudinal sNfL data were available for 60 pwMS. We used adjusted partial correlations and multiple linear regression to identify predictors of lesion load and brain volume.

Results: CPV (p = 0.002) and sNfL (p < 0.001) were significantly higher in pwMS compared to healthy controls. Cross-sectional regression showed CPV was independently linked to reduced brain volume and higher lesion load (β = -0.02; p < 0.001 and β = 1.55; p < 0.001). In longitudinal regression, only sNfL remained a significant predictor of brain atrophy (β =1.33; p = 0.003), not CPV.

Conclusion: Although both CPV and sNfL correlate with MRI signs of brain damage in pwMS, longitudinal analysis identified sNfL as the sole marker linked to more pronounced brain atrophy.

P34: The importance of routine cognitive assessment in patients with multiple sclerosis: A real-world study

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Introduction: Cognitive impairment is a frequent and disabling symptom in multiple sclerosis (MS) with information processing speed being one of the most affected domains. Various factors, such as age at disease onset, disability progression, and treatment may influence cognitive performance. Understanding these factors can help to refine treatment strategies and improve the quality of care for MS patients experiencing cognitive decline.

Objective: The aim was to analyze the impact of disease-specific factors on cognitive performance in a real-world study.

Method: Patients who were treated at the MS clinic of the Medical University of Innsbruck and who had at least 1 assessment using the Symbol Digit Modalities Test (SDMT)—a widely used, sensitive measure for cognitive performance particularly for information processing speed—were eligible for inclusion. SDMT z-scores adjusted for age, sex, and educational level were used. Based on the age of disease onset, patients were categorized into early-onset MS (< 50 years) and late-onset MS (≥ 50 years). Disability was assessed by the Expanded Disability Status Scale (EDSS) score. Disease-modifying treatments (DMT) were categorized as moderate-efficacy (ME) and high-efficacy (HE) DMT. Statistical analyses were performed by multivariable linear regression.

Results: X: A total of 583 MS patients (median age 43 years [25th–75th percentile 34–53], 421 [72 %] female, 522 [90 %] relapsing-remitting [RRMS] and 61 [10 %] secondary progressive disease course [SPMS], disease duration 10 [5–18] years) were included in the study. Thirty (5 %) patients had late disease onset. The median EDSS score was 2 (0–2.5). While 230 (39 %) patients were treated with ME-DMT, 285 (49 %) patients were on HE-DMT and 68 (12 %) did not receive any DMT. Regression analysis revealed that disease duration (β = -0.02, p = 0.006) and higher EDSS (β = -0.16, p < 0.001) were associated with lower SMDT z-score, while late disease onset and type of DMT had no statistically significant impact. A total of 265 patients had a follow-up visit after a median time interval of 7 (6–13) months. For the change (Δ) in SDMT z-score, only the EDSS score at first SDMT was predictive (β = -0.06, p = 0.017), while disease onset, disease duration, type of DMT, and the occurrence of relapses were not. Of 203 patients with stable EDSS, 78 (38 %) experienced a decrease in SDMT z-scores. In these patients, only higher EDSS score at baseline predicted SDMT deterioration (β= -0.08, p = 0.001). An EDSS score > 4 was identified as useful cut-off.

Conclusion: Screening of cognitive performance by SDMT should be routinely performed, especially in patients with higher disability, as cognitive deterioration can occur even in patients who are assumed stable based on the EDSS.

P35: Risikofaktoren für einen kurzfristigen Knochenabbau bei Multipler Sklerose

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Hintergrund: Eine verringerte Knochenmasse und ein erhöhtes Osteoporoserisiko sind häufig bei Menschen mit Multipler Sklerose (MS). Ziel der Studie war die Ermittlung von Risikofaktoren für kurzfristigen Knochenabbau bei MS.
Fragestellung und Methode: Diese prospektive Studie umfasste 159 MS-Patient*innen (im Alter von 18–65 Jahren), von denen 139 den 2-Jahres-Follow-up abschlossen. Zu den Ausgangsdaten gehörten demografische Daten, Body-Mass-Index (BMI), körperliche Aktivität, Rauchen, Menopausen-Status, 25-Hydroxy-Vitamin-D-Spiegel und Glukokortikoideinnahme. Die Knochenmineraldichte (BMD) wurde zu Beginn der Studie und nach 2 Jahren mittels Dual-Energy-Röntgen-Absorptiometrie (DXA) an der Lendenwirbelsäule und Hüfte gemessen. Die Verschlechterung der Behinderung wurde anhand der Expanded Disability Status Scale (EDSS) bewertet.
Ergebnisse: Während der 2-Jahres-Follow-up-Periode wurde ein signifikanter BMD-Verlust in der Hüfte beobachtet (Baseline g/cm2: Median 0,898; IQR 0,808–1,014; 2-Jahres-Follow-up: 0,882; 0,784–1,01; $p < 0,001$), nicht aber in der Lendenwirbelsäule. Insgesamt kam es bei 103 Patient*innen (74,6 %) zu einem Hüft-BMD-Verlust, mit einer medianen Abnahme von 3,5 %. Die Regressionsanalyse ergab eine Verschlechterung der Behinderung als signifikanten Prädiktor für den Hüftknochenverlust (Odds Ratio: 14, $p < 0,012$). MS-Patient*innen, die während des Nachbeobachtungszeitraums Frakturen erlitten hatten, wiesen signifikant niedrigere BMD-Werte und einen höheren EDSS auf und waren älter als diejenigen, die keine Frakturen erlitten hatten.
Zusammenfassung: Behinderungsprogression wurde als Risikofaktor für einen beschleunigten BMD-Verlust identifiziert. Diese Ergebnisse unterstreichen die Notwendigkeit einer aktiven Überwachung von MS-Patient*innen mit Behinderungsverschlechterung, um einen Knochenverlust zu verhindern und damit das Frakturrisiko zu verringern.

P36: Acute headache treatment in idiopathic intracranial hypertension: Treating to the phenotype?

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Background: Effective acute headache treatment is essential for improving quality of life for people with idiopathic intracranial hypertension (pwIIH), with experts recommending "treating to the phenotype" despite limited data.
Method: This retrospective analysis used standardized headache diaries of pwIIH from the Vienna Idiopathic Intracranial Hypertension (VIIH) database from July 1 2021 to June 30 2023. We analyzed 3 classes of acute medication (acetaminophen [APAP], nonsteroidal anti-inflammatory drugs [NSAIDs], and triptans) with NSAIDs as reference, and 9 substances combining different doses and formulations by their generic names with ibuprofen as reference. Headache attacks were classified according to ICHD-3 as migraine without/with aura (M), tension-type (TTH), or other (O). We used 2-level nested logistic regression models to analyze odds ratio (OR) of patient-reported good response, adjusting for covariance within individual pwIIH and concurrent medication with propensity weighting for age, sex, and headache severity.
Results: We examined 35640 medication-outcome pairs from 23507 headache attacks (M: 45.3 %, TTH: 21.1 %, O: 33.6 %) in 156 patients (89.7 % female, mean age 32.9 years). NSAIDs

were most frequently used across all headache types (M: 60.5 %, TTH: 69.8 %, O: 70.7 %), followed by APAP (M: 21.5 %, TTH: 21.1 %, O: 27.7 %) and triptans (M: 18 %, TTH: 10.1 %, O: 12.8 %). Compared to NSAIDs, triptans were most effective across all headache types (OR for M: 4.8 [CI 3.9–6.1], TTH: OR 2.9 [CI 1.8–4.3], O: OR 3.1 [CI 2.2–4.3]), while APAP was less effective for M (OR 0.81 [CI 0.74–0.90]) but similarly effective in TTH and O. In M, eletriptan and zolmitriptan (OR 6.0 and 5.8) were slightly more effective than sumatriptan (OR 5.0).

Conclusion: Triptans are more effective than NSAIDs and APAP in the acute management of headaches in pwIIH, particularly—but not exclusively—for migraine-type attacks. These findings support the preferential use of triptans, questioning the "treating to the phenotype" approach.

P37: Calcitonin Gene-Related Peptide (CGRP) und andere Neuropeptide bei akuten und postakuten Schlaganfallpatient:innen

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Hintergrund: Das Calcitonin Gene-Related Peptide (CGRP) gilt als eines der stärksten gefäßerweiternden Neuropeptide und spielt eine entscheidende Rolle in der Pathophysiologie der Migräne. In den letzten Jahren hat sich die gezielte Blockade des CGRP-Signalwegs durch monoklonale Antikörper oder Gepante als vielversprechender Ansatz in der akuten und prophylaktischen Migränetherapie bewiesen. Diese neuartigen Therapieansätze sind verglichen zu traditionellen oralen Prophylaxetherapien nebenwirkunksärmer und effektiver. Im Tiermodell konnte allerdings gezeigt werden, dass eine Blockade der CGRP-Rezeptoren zu einem größeren Infarktareal im Schlaganfallmodell führt. Darüber hinaus entwickelten Mäuse mit blockierten CGRP-Rezeptoren im TIA-Modell (transitorische ischämische Attacke) einen manifesten Schlaganfall.

Das Pituitary Adenylate Cyclase-Activating Polypeptide (PACAP), das ebenfalls eine bedeutende Rolle bei der Gefäßerweiterung spielt, wird derzeit in Phase-II-Studien als potenzielles Ziel für die Migräneprävention untersucht und könnte in naher Zukunft eine Rolle in der Migräneprophylaxetherapie spielen.

Die endotheliale Dysfunktion wird sowohl mit dem Schlaganfall als auch mit der Migräne in Verbindung gebracht. Angiopoietin-1 (Ang-1), Angiopoietin-2 (Ang-2) und lösliches Tie-2 (sTie-2) stellten sich dabei als zentrale Akteure in der endothelialen Pathophysiologie heraus und könnten möglicherweise eine Verbindung zwischen Migräne und Schlaganfall aufzeigen.

Fragestellung: Aufbau einer Biodatenbank für CGRP, PACAP, Ang-1, Ang-2 und sTie-2 bei Patient*innen mit akutem und postakutem ischämischem Schlaganfall und Untersuchung einer Dynamik dieser Peptide zu den 3 Zeitpunkten.

Methoden: Diese prospektive, monozentrische Querschnittsstudie schließt Patient*innen mit akutem ischämischen Schlaganfall seit 02.05.2023 ein. Eine Berechnung der Stichprobengröße ergab n = 163 bei einer kleinen Effektstärke, einem alpha = 0,05 und einer Power = 0,80. Die Diagnose des ischämischen Schlaganfalls wird durch eine/n Fachärztin/Facharzt für Neurologie und mittels neuroradiologischer Bildgebung gestellt. Bei allen eingeschlossenen Patient*innen werden 3 aufeinanderfolgende Blutabnahmen aus der Kubitalvene durchgeführt: Diagnose in der Notaufnahme (T1), am darauffolgenden Tag des Schlaganfalls (T2) und nach 3 Monaten (T90). Bei Patient*innen, die für eine Thrombektomie infrage kommen, wird eine vierte Blutentnahme aus der betroffenen Hirnarterie im Rahmen der Thrombektomie durchgeführt. Die Proben werden nach einem vordefinierten Protokoll verarbeitet und bei –80 °C gelagert, bevor die Konzentrationen von CGRP, PACAP, Ang-1, Ang-2 und sTie-2 mittels spezifischer ELISA-Kits gemessen werden.

Ergebnisse: Bisher wurden Blutproben von 70 Proband*innen verarbeitet, 47 erfüllen die Ein- und Ausschlusskriterien der Studie (Frauen = 21, Männer = 26). Von 15 Proband*innen liegen bereits preliminiäre Ergebnisse der CGRP-Spiegel zu allen 3 Zeitpunkten (T1, T2 und T90) vor, von 2 zusätzlich aus der betroffenen Hirnarterie. Die CGRP-Spiegel zeigen eine große interindividuelle Varianz (0,00 pg/ml – 305,01 pg/ml). Zu den Zeitpunkten T1, T2 und T90 konnte keine statistisch signifikante Dynamik ermittelt werden. Ebenso gibt es keinen signifikanten Unterschied zwischen venösen und intraateriellen CGRP-Spiegeln. Es wurde kein signifikanter Unterschied bei den Werten zwischen Frauen und Männern ermittelt. ▶

Zusammenfassung: Insgesamt kann gesagt werden, dass die longitudinalen CGRP-Spiegel bei der derzeitigen geringen Proband*innenanzahl (n = 15) einer statistisch nichtsignifikanten Dynamik unterliegen. Um eine etwaige Dynamik erkennen zu können, wird die Rekrutierung bis zur berechneten Proband*innenanzahl fortgesetzt. Interindividuell sind die CGRP-Spiegel sehr unterschiedlich; dies ist bereits in der Literatur beschrieben.

P38: Migräne-Patient*innen mit kraniellen autonomen Symptomen und deren Ansprechen auf eine Therapie mit monoklonalen Antikörpern gegen Calcitonin Gene-Related Peptide (Rezeptoren) – eine Real-World-Studie

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Hintergrund: Migräne als sowohl häufige als auch komplexe Erkrankung ist durch eine Reihe von heterogenen klinischen Merkmalen gekennzeichnet, einschließlich Variationen in der Dauer der Attacken, Auftreten einer Aura, Schmerzintensität und begleitender Symptome. Zu letzteren können häufigere Phänomene wie Foto- oder Phonophobie, Übelkeit und Erbrechen oder weniger gängige wie Allodynie, Stimmungsschwankungen oder kraniale autonome Symptome (CAS) gehören. CAS, charakterisiert durch konjunktivale Injektion, Tränenfluss, nasale Kongestion, Rhinorrhö, Lidödem, Miosis und Ptosis, sind am häufigsten mit trigeminalen autonomen Kopfschmerzerkrankungen (TACs) verbunden, können jedoch auch bei Migräne auftreten. Das Calcitonin Gene-Related Peptide (CGRP) spielt eine Schlüsselrolle in der Migräne-Pathophysiologie und -behandlung. CGRP-Rezeptoren sind im Trigeminusganglion, einer kritischen Region in der Entwicklung von Gesichtsschmerzen und CAS, in hoher Zahl nachweisbar. Neueste Studien zeigten bereits eine größere Reduktion der monatlichen Kopfschmerztage (MHD) bei Patient*innen mit CAS-Symptomen, die mit monoklonalen Antikörpern (mAb) gegen CGRP(R) behandelt wurden, im Vergleich zu denen ohne CAS. Diese Hypothese wird auch dadurch unterstützt, dass bei CAS+ Patient*innen höhere CGRP-Level im Blut gemessen werden konnten und diese besser auf Triptane ansprachen. Triptanresponder haben in Studien ein besseres Ansprechen auf eine Therapie mit CGRP(R) mAbs gezeigt als jene mit unzureichendem Ansprechen auf Triptane.

Fragestellung: Ziel dieser Studie ist es zu evaluieren, ob bei Patient*innen unter mAb-Therapie ein signifikanter Unterschied zwischen CAS+ und CAS– in Bezug auf die Reduktion der MHD besteht.

Methoden: Die Teilnehmer*innen wurden durch das Österreichische Migräneregister (AMRC), einer multizentrischen österreichweiten Fragebogenstudie, die Real-World-Daten in der Anwendung von CGRP-mAbs sammelt, gescreent. Jene, die bei der Frage nach „Augentränen, Augenrötung, laufende oder verstopfte Nase, hängendes Lid" angaben, „leicht, mittelmäßig oder schwer" während einer Migräneattacke betroffen zu sein, wurden für ein standardisiertes Telefoninterview kontaktiert. Anhand der CAPS-Skala, unterteilt in mild (1–2 Punkte), moderat (3–4), schwer (5–6) und sehr schwer (> 7), wird die Intensität dieser Symptomatik quantifiziert. Mittels G*Power wurde die Fallzahl auf 100 CAS+ gerechnet, die mit 100 CAS– Kontrollgruppen in den Parametern Geschlecht, MHD, Alter und Vortherapien gematched werden.

Ergebnisse: Bisher konnten zwischen dem 25. Oktober 2024 und 7. Jänner 2025 23 Innsbrucker Teilnehmer*innen, davon 20 Frauen und 3 Männer, erfolgreich telefonisch kontaktiert werden. Die CAS verteilten sich bei der Baseline (vor mAb-Therapie) wie folgt: mild n = 8, moderat n = 5, schwer n = 4, sehr schwer n = 2. Beim Follow-up nach mindestens 3 Monaten Abstand zur Baseline waren bei n = 6 keine CAS mehr nachweisbar, bei n = 10 mild, n = 2 moderat und bei n = 1 noch schwer nachweisbar. Bei 4 der im Fragebogen positiven Teilnehmer*innen zeigten sich nach standardisiertem CAPS-Interview keine CAS.

Zusammenfassung: Insgesamt lässt sich in Bezug auf die Ausprägung der CAS ein positives Ansprechen auf die prophylaktische Therapie mit den mAbs erkennen. Für die Berechnung der Reduktion der MHD unter mAb-Therapie bei CAS+ vs. CAS– Proband*innen ist der Einschluss weiterer Proband*innen notwendig.

P39: Serum neurofilament light chain and glial fibrillary acidic protein levels in idiopathic intracranial hypertension: An exploratory study

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Background: Idiopathic intracranial hypertension (IIH) is a systemic disorder marked by increased intracranial pressure carrying the risk of blindness due to optic nerve damage. Serum neurofilament light chain (sNfL) and glial fibrillary acidic protein (GFAP) are emerging biomarkers of axonal damage and astrocytic activation, respectively. However, their roles within the framework of IIH remain underexplored.

Objective: We aimed to evaluate the sNfL and GFAP levels in people with newly diagnosed IIH (pwIIH) and investigate their potential association with ophthalmological outcomes.

Method: From an ongoing prospective observational study including pwIIH, sNfL and GFAP levels were measured at baseline using single molecule array (Simoa) technology, and analyzed as z-scores adjusting for age, BMI and—for GFAP—sex. The ophthalmological outcomes included papilledema degree, visual outcomes (visual acuity, visual field), optical coherence tomography (peripapillary retinal nerve fiber layer [pRNFL] thickness, and ganglion cell layer [GCL] volume), and transbulbar sonography (arachnoid optic nerve sheath diameter [AONSD]).

Results: We included 23 pwIIH (mean age 34.3 years [SD 8.1], 95.7 % female, median cerebrospinal fluid [CSF] opening pressure 33.0 cmCSF [IQR 26.9–35.4], median body mass index (BMI) 35.7kg/m2 [IQR 31.1–43.3]). Mean sNfL and GFAP z-scores at baseline were 1.0 (1.0) and 0.5 (1.4), respectively. SNfL z-scores at baseline exhibited a non-significant positive correlation with the GCL volume of the worse eye ($r = 0.38$, $p = 0.079$), whereas GFAP z-scores showed no correlation with any ophthalmological outcomes. Additionally, neither sNfL nor GFAP z-scores correlated with the CSF opening pressure.

Conclusion: SNfL might be associated with GCL volume, an established sensitive marker of optic nerve damage, suggesting its potential relevance in reflecting neuroaxonal damage in IIH. In contrast, GFAP does not appear to correlate with any ophthalmological outcomes. Further studies with larger cohorts are warranted to determine whether sNfL could serve as a prognostic biomarker in IIH. Given that IIH entails impaired CSF homeostasis, the lack of correlation between serum biomarkers and outcomes might be attributable to impaired outflow of biomarkers from the CSF into the bloodstream, a hypothesis we aim to explore in future investigations.

P40: Postoperative Polyradikulitis nach Lipofilling

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Hintergrund: Das Guillain-Barré-Syndrom (GBS) ist eine akute immunvermittelte Polyradikulitis und eine der häufigsten Ursachen für akute schlaffe Lähmungen weltweit (Inzidenz 1–2: 100.000 Patientenjahre). Neben postinfektiösem und postvakzinalem Auftreten werden auch chirurgische Eingriffe binnen 6 Wochen vor Symptombeginn als Auslöser berichtet. Die genaue Pathophysiologie einer – eher selten auftretenden – postoperativen Polyradikulitis ist unklar; epidemiologische und retrospektive Studien zeigen jedoch einen klaren Zusammenhang.

Fragestellung: Wie unterscheidet sich eine postoperative von einer postinfektiösen Polyradikulitis?

Methode: Fallbericht

Ergebnisse/Fallbericht: Ein bis dato gesunder 49-jähriger Patient wurde wegen seit 4 Tagen bestehender progredienter, symmetrisch aufsteigender distaler Missempfindungen an OE und UE bds. aufgenommen. 13 Tage zuvor waren diverse ästhetisch-plastische Eingriffe im Rahmen einer prolongierten Operation in Vollnarkose in der Türkei erfolgt. Es lag kein Befundbericht vor, laut Patient wurde eine Liposuktion des Abdomens mit Lipofilling in Waden/Knöchel sowie im Gesicht und Lidraffung durchgeführt.

Klinisch neurologisch zeigten sich sensomotorische distale Ausfälle an OE und UE bds. mit einem sensibel-aktaktischen Gangbild, bilateralen Fußparesen und abgeschwächtem ASR bds. Es bestanden massive neuropathische Schmerzen ▶

im Bereich der OE und UE. Liquordiagnostisch zeigte sich zytoalbuminäre Dissoziation. Die NLG dokumentierte an den motorischen Nerven der OE und UE diskrete Zeichen einer Demyelinisierung sowie einen kompletten Ausfall der F-Wellen der UE und verlängerte minimale F-Wellen-Latenz der OE. Unmittelbar wurde ein gewichtsadaptierter IVIG-Zyklus begonnen. Dennoch kam es zur raschen Verschlechterung mit hochgradig beinbetonter Tetraparese, Bulbärsyndrom mit ausgeprägter Dysphagie und Fazialisparese links sowie aggravierter Schmerzsymptomatik. Aufgrund respiratorischer Insuffizienz und Aspirationspneumonie erfolgte am fünften Tag des Aufenthaltes die Verlegung auf die Intensivstation mit invasiver Beatmung für 10 Tage. Nach Infektbehandlung kam es während des ICU-Aufenthaltes ohne weitere Immuntherapie zur Besserung von Atmung und Motorik, sodass am Tag 20 die Rückverlegung an die neurologische IMC erfolgte. Unter intensiver Frührehabilitation kam es zu einer langsamen Besserung mit oralem Kostaufbau, Reduktion der Analgetika und Mobilisation im Stehen und Gehen mit Begleitperson. Am Tag 65 erfolgte die Entlassung zum neurologischen Anschlussheilverfahren. Es bestand eine mäßige Paraparese, Gehen war mit Begleitperson einige Ganglängen möglich. Auch die polypragmatische analgetische und schmerzmodulierende Therapie (Metamizol, Tramadol, Pregabalin, Duloxetin und zuletzt Ambroxol topisch) wurde reduziert. Elektromyografisch fanden sich lediglich im M. tibialis diskrete Zeichen einer floriden Denervierung; die Gangliosidantikörper waren negativ.

Zusammenfassung: Die akute Polyradikulitis ist eine seltene postoperative Komplikation mit einer vermutlich durch Gewebszerfallsprodukte und Entzündungsmediatoren bedingten Autoimmunreaktion. Es wird eine komplexe Interaktion aus perioperativem Stress, operativem Verfahren selbst, Komorbidität, Anästhesie und genetischen Faktoren angenommen. Obwohl unser Fall eine AIDP (akute inflammatorische demyelinisierende Polyneuropathie) beschreibt, finden sich in der Literatur Hinweise auf eine höhere Inzidenz von axonalem Schädigungsmuster beim postoperativen GBS. Zudem zeichnet sich die postoperative Polyradikulitis durch häufig raschere Progression, schweren Verlauf und respiratorische Insuffizienz sowie etwas häufigeres Auftreten bei Männern aus. In Fallserien werden auch kürzere Latenzen zwischen neurologischem Symptombeginn und auslösendem Ereignis beschrieben; gerade bei postoperativer Analgosedierung/Beatmung kann dies die Diagnosestellung erschweren. Diagnose und Therapie folgen den klassischen Regeln der akuten Polyradikulitis. Eine zeitnahe Immuntherapie mit IVIG oder Plasmapherese verbessert Symptome und Prognose.

P41: Slow-channel congenital myasthenic syndrome due to the novel variant c.1396G>A in CHRNA1 that responds favorably to 3,4 DAP

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Objective: Mutations in CHRNA1 are responsible for postsynaptic CMS and occur either as slow channel syndrome or fast channel syndrome. Slow channel CMS due to CHRNA1 variants responds favorably to pyridostigmine. A patient with slow-channel CMS due to a new CHRNA1 variant that responds favorably to 3,4-diaminopyridine (3,4-DAP) has not yet been reported.

Case Report: The patient is a 36-year-old woman who was diagnosed with non-specific CMS at the age of 1 year when she presented clinically with signs of somnolence, weakness, and facial dysmorphism. She later also developed limb weakness, with the upper limbs being more severely affected. Heat, low humidity, late menstruation, high fever, and stress aggravated the muscle weakness. Only at the age of 17 was pyridostigmine started, which partially improved the muscle weakness. The diagnosis was genetically confirmed when the new, heterozygous variant NM_001039523:c.1396G>A in CHRNA1 p.(Gly466Arg) was detected at the age of 30. Since then, 3,4-DAP was administered, which further improved the muscle weakness.

Conlcusion: CHRNA1-associated slow-channel CMS may respond favorably not only to pyridostigmine but also to the additional administration of 3,4-DAP. Patients with CHRNA1-associated CMS can live for years without treatment, especially in early life; CMS should be diagnosed without delay to avoid putting people at risk of receiving medication that could potentially worsen their phenotype

P42: Leigh syndrome due to the compound heterozygous variants c.1162A>C/c.1138G>C in NDUFV1

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Background: Early-onset Leigh syndrome is usually a genetically and phenotypically heterogeneous, severe, rapidly progressive mitochondrial disorder with a fatal outcome. Leigh syndrome is genetically heterogeneous as it is based on mutations in mtDNA or nDNA genes, which mostly encode subunits of respiratory chain complexes or assembly factors. It is phenotypically heterogeneous because it is genetically heterogeneous and due to the peculiarities of mitochondrial genetics. One of the more than 100 mutated genes responsible for Leigh syndrome is NDUFV1. Here, we present an infant with Leigh syndrome who suffers from a novel heterozygous variant of NDUFV1, which is phenotypically characterized by a number of previously unknown features.
Case report: The patient is a 4-month-old girl with Leigh syndrome due to the compound heterozygous variants c.1162+4A>C (previously described, inherited from the mother) and c.1138G>C (novel, inherited from the father) in NDUFV1. The mutation c.1162+4A>C is a non-canonical splice site variant that has been demonstrated to result in loss of function. Bioinformatic analysis supports that the missense variant c. 1138G>C has a deleterious effect on protein structure or function. The mutations manifested phenotypically with typical cerebral lesions on imaging, developmental delay, cognitive decline, epileptiform discharges in the electroencephalography without seizures, AV block II, agenesis of a subclavian vein, right heart failure, patent foramen ovale, pulmonary hypertension, hypoaldosteronism, and abdominal hernias. Within 5 weeks of hospitalization, the disease took a progressive course, and the patient died of infectious complications despite maximum treatment.
Conclusion: This case shows that the described new heterozygous variant in NDUFV1 can occur with previously undescribed phenotypic features. It is important to diagnose mitochondrial disorders due to NDUFV1 mutations early in order not to miss the time for appropriate symptomatic treatment.

P43: Clinical and demographic characterization of patients with 5q-associated spinal muscular atrophy in Austria:The SMAustria project (part A)

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Introduction: Spinal muscular atrophy (SMA) is a genetic motor neuron disease caused by homozygous deletions or (likely) pathogenic variants in the SMN1 gene associated with loss-of-function of the SMN protein. The resulting deficiency of the survival motor neuron (SMN) protein leads to degeneration of lower motor neurons associated with progressive weakness and muscle wasting. Disease severity varies depending on the SMN2 copy number. The development and approval of disease-modifying therapies (DMT) in recent years has fundamentally transformed the management of SMA patients and may contribute to a change of the demographic and phenotypical landscape of the disease. ▶

Aim and Method: Part A of this study aims to provide a clinical and demographic characterization of patients with SMA in Austria. In this retrospective nationwide cohort study, patients with 5q-associated SMA treated at pediatric and adult neuromuscular centers across Austria were included. Clinical and demographic data were extracted from the hospitals' patient data management system.

Results: A total of 60 pediatric and 118 adult patients were included in this study. Females accounted for 50.0 % of the cohort. Median age at onset was 1.5 years (IQR 1.0–6.0) in adults and 0.6 years (IQR 0.2–1.4) in pediatric patients, respectively. 18.3 % of pediatric patients were presymptomatic at inclusion. In adult patients, median age at genetic diagnosis was 27.8 years (IQR 16.6–37.). Median age at genetic diagnosis in pediatric patients was 0.5 years (IQR 0.0–2.5). SMA type III was most commonly diagnosed in adult SMA patients (55.1 %), while pediatric patients most commonly suffered from SMA type I (35.0 %). 88.1 % of adult and 96.7 % of pediatric patients received DMT. Eighty-seven and 34 adult patients received risdiplam and nusinersen, respectively. In the pediatric cohort, 27 patients received risdiplam, 23 patients received nusinersen and 25 patients received onasemnogene abeparvovec. According to the impression of treating physicians, a clinical stabilization or improvement of motor functions were observed in most patients with DMT.

Summary: This nationwide study provides comprehensive clinical and demographic data on a large cohort of pediatric and adult SMA patients. Future analyses (parts B-D of the SMAustria project) will evaluate SMA prevalence in Austria, the frequency of SMA-associated comorbidities, and treatment adherence.

Acknowledgement: This study was financially supported by Biogen, Novartis, and Roche.

P44: Development of a monitoring tool for therapy management in CIDP

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Background: Chronic inflammatory demyelinating polyneuropathy (CIDP) is a rare immune-mediated neuropathy characterized by symmetrical peripheral nerve involvement. There are several approved therapies, as well as off-label options, currently available, and numerous new drugs are under investigation. Standardized guidelines for modifying or escalating therapy in patients with inadequate therapy response do not currently exist. To eliminate the risk of misdiagnosing treatment responses, it is essential to ensure consistent and standardized documentation of clinical progress.

Research question: What are the key considerations based on current literature regarding the development of a symptom monitoring tool for CIDP?

Method: Based on current literature, we designed a documentation form that summarizes the most important clinical scales for CIDP.

Results: This monitoring tool for therapy management in CIDP consists of part A) current therapy (indication of immunotherapy, dose and application interval); part B) medical history, examination findings, and therapy effects; Part C) progression of the findings; and part D) further therapeutic procedure.

Conclusion: This tool provides clear and practicable documentation for everyday clinical practice. By means of a simplified and uniform protocol, clinicians can more effectively assess patient progress, tailor interventions, and ultimately improve the quality of care for patients with CIDP.

Acknowledgement: This study was financially supported by Kedrion Biopharma

P45: High-definition profiling of skin microenvironment in post-COVID small fiber neuropathy

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Small fiber neuropathy (SFN) is characterized by the loss of thinly myelinated A-delta fibers and unmyelinated C-fibers, resulting in sensory deficits and dysautonomia. SFN emerged as one major neurological complication of post-COVID. Here we leverage a large cohort of patients with post-COVID to systematically study skin biopsies using spatial biology methods to focus on the role of Schwann and immune cells as critical determinants of nerve fiber regeneration. Methodologically, we employed immunofluorescence and high-definition spatial transcriptomics (10X Genomics) to integrate nerve fiber density with cell-type specific transcriptional programs. To this end, the cohort comprises 34 females and 13 males with older individuals (median age 41 years, p = 0.003, Mann-Whitney) and those with a non-length-dependent SFN demonstrating more pronounced nerve fiber loss (p = 0.0172, Kruskal-Wallis). Subsets had overlapping myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS, n = 15) and postural orthostatic tachycardia syndrome (POTS, n = 15). Overall, autoimmune and connective tissue disorders comprised major comorbidities (n = 37). Our preliminary analysis of Visium HD datasets (n = 13) identified 26 distinct clusters using dimensionality reduction based on the Louvain algorithm in post-COVID skin biopsies compared to 16 in control skin. Using marker genes to define associated cell types revealed activation of cellular responses to cytokine stimuli, dermal macrophage activation, and upregulation of oxidative stress pathways. Several upregulated genes, e.g., COL1A2, have been linked to inflammatory conditions, including Ehlers-Danlos syndrome, and have also been observed in the context of COVID-19. In contrast, we observed significant downregulation of genes critical for maintaining skin homeostasis and structural integrity. Ultimately, we identified varied immature states of neural crest-derived cells, including Schwann cells, at the dermal-epidermal junction, indicative of dynamic cellular activity, which is currently being investigated in the context of SFN. Taken together, our findings suggest that cellular and molecular changes in the skin microenvironment play a critical role in the pathophysiology of SFN.

P46: Real-world reduction in oral corticosteroid utilization at 1 year following efgartigimod initiation

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Background: Efgartigimod (EFG) is a human IgG1 Fc fragment engineered to bind to the FcRn receptor on endothelial cells, leading to increased degradation of IgG (including pathological IgG) in the lysosome. Efgartigimod was approved for the treatment of anti-AChR antibody–positive gMG in 2021.
Research question: To evaluate oral corticosteroid (OCS) usage at 1 year following EFG initiation. Method: Patients with gMG using OCS pre-EFG initiation were identified from a United States medical and pharmacy claims database (based on information li- ▶

censed from IQVIA: Longitudinal Access and Adjudication Data [LAAD] for the period April 2016–December 2023, reflecting estimates of real-world activity [all rights reserved]). Mean (standard deviation [SD]) average daily dose (ADD) of OCS was evaluated during the 3 months prior to, and at 6- and 12-months post-EFG initiation. To assess outcomes, de-identified Myasthenia Gravis Activities of Daily Living (MG-ADL) data collected in the "My VYVGART Path" patient support program was tokenized and integrated into the primary dataset.

Results: A total of 169 adults (aged ≥ 18 years) who were using chronic OCS pre-EFG initiation initiated EFG by December 31 2022 and continued EFG for at least 12 months were included in the analysis. At 6 and 12 months post-EFG, respectively, 31 (18 %) and 45 (27 %) patients had no OCS usage. Overall mean (SD) OCS ADD was significantly reduced at 6 months (13.2 [13.9] mg/day, $p < 0.001$), and at 12 months (10.2 [12.1] mg/day, $p < 0.001$) post-EFG initiation compared with baseline (17.2 [13.7] mg/day). Among a subset of 72 patients (43 %) who had both pre- and post-EFG MG-ADL scores available, best follow-up mean (SD) MG-ADL was significantly improved (from 8.3 [3.7] to 3.4 [2.8], $p < 0.001$).

Summary: The significant reduction of OCS usage observed at 6 months post-EFG initiation was retained at 12 months, while demonstrating MG-ADL response expected from EFG treatment.

P47: Efficacy and safety of efgartigimod PH20 subcutaneous in chronic inflammatory demyelinating polyneuropathy: Results of ADHERE/ADHERE+

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Background: Efgartigimod, a human immunoglobulin G (IgG)1 antibody Fc fragment, blocks the neonatal Fc receptor, decreasing IgG recycling and reducing pathogenic IgG autoantibody levels. Research question: To assess the efficacy and safety of efgartigimod PH20 subcutaneous (SC; coformulated with recombinant human hyaluronidase PH20) in chronic inflammatory demyelinating polyneuropathy (CIDP) in the multi-stage, double-blinded, placebo-controlled ADHERE and ongoing open-label extension ADHERE+ studies.

Method: Enrolled participants with CIDP

(off treatment or on standard treatments withdrawn during run-in) had active disease and received once-weekly (QW) efgartigimod PH20 SC 1000mg (stage A). Responders were randomized (1:1) to QW efgartigimod PH20 SC 1000mg or placebo (stage B). Participants with clinical deterioration in stage B or those who completed ADHERE could enter the ongoing, open-label extension ADHERE+ trial (QW efgartigimod PH20 SC 1000mg). Primary outcomes were confirmed evidence of clinical improvement (ECI) (stage A), relapse risk (stage B), and safety (ADHERE+).

Results: In stage A, 214/322 (66.5 %) participants demonstrated confirmed ECI. In stage B, efgartigimod significantly reduced relapse risk (hazard ratio: 0.394 [95% confidence interval 0.253–0.614]) vs. placebo (p = 0.00004). This reduction was observed regardless of prior CIDP therapy. 99 % of eligible participants entered ADHERE+. The safety profile of efgartigimod was consistent over 137.42 total patient-years of follow-up for ADHERE+. Most treatment-emergent adverse events were mild/moderate; the incidence/severity did not increase in ADHERE+.

Summary: ADHERE demonstrated effectiveness of efgartigimod PH20 SC in reducing relapse risk. The safety profile of efgartigimod PH20 SC was similar between ADHERE and ADHERE+ and was consistent with the previously demonstrated safety profile of efgartigimod.

P48: A deeper look into seronegative myasthenia gravis (SNMG): Clinical, immunological, and genetic insights from an Austrian patient cohort

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Background: Myasthenia gravis (MG) encompasses a variety of disorders characterized by dysfunction of the neuromuscular junction. Although most cases are associated with autoantibodies (Abs) against postsynaptic components, around 10 % remain unresolved and are thus classified as seronegative (SNMG). The etiology of SNMG is diverse, ranging from different autoimmune mechanisms without detectable Abs to genetic mimics as potential causes.

Aim: The aim of this project is to provide an in-depth clinical, immunological and genetic characterization of SNMG patients using an extended Ab screening with cell-based assays (CBAs) and whole-exome sequencing (WES) to identify underlying genetic causes, especially congenital myasthenic syndromes (CMS).

Aim: The aim of this project is to provide an in-depth clinical, immunological and genetic characterization of SNMG patients using an extended Ab screening with cell-based assays (CBAs) and whole-exome sequencing (WES) to identify underlying genetic causes, especially congenital myasthenic syndromes (CMS).

Method: Within the framework of this Austrian multi-center study, a comprehensive CBA-based screening for Abs against clustered AChR, MuSK, and LRP4, as well as WES was conducted. Sixty patients were recruited at the Departments of Neurology of the Medical Universities of Vienna (n = 39), Innsbruck (n = 12), and Graz (n = 9). All participants had no previously identified anti-acetylcholine receptor (AChR) or anti-muscle-specific kinase (MuSK) antibodies through clinical routine diagnostic testing by RIA.

Results: Forty-three of the 60 patients (71.7 %) were female with a mean onset age of 38.5 (95% CI, 35.0–43.3) years. Most patients (61.7 %) presented with ocular and 25 % presented with generalized symptoms at onset, whereas 33.3 % showed ocular and 53.3 % generalized manifestations at the time of recruitment. Treatment at that time included pyridostigmine (n = 37), steroids (n = 26), non-steroidal immunosuppressants (n = 26), rituximab (n = 3), eculizumab (n = 2), and plasma exchange or intravenous immunoglobulins (n = 6). 39.7 % of patients underwent thymectomy and the family history was positive in 11.7 % of cases. Mean MG-ADL scores at inclusion were 4.8 (95% CI, 3.8–5.8). Through CBA testing, Abs against clustered AChR and MuSK were found in 10 % (n = 6) and 6.7 % (n = 4) of subjects, respectively. Among the remaining 50 patients, 7 patients (14 %) had CMS with genetic variants in CHRNE ▶

(n = 4) and RAPSN (n = 3). Furthermore, genetic findings of uncertain clinical significance were reported in 4 cases, including the genes RAPSN, DOK7, DPAGT1, and CACNA1S.

Conclusion and outlook: In this study, we present a comprehensive immunological, genetic and clinical characterization of an Austrian SNMG cohort. Overall, our findings provide evidence supporting the implementation of CBA in routine diagnostics, which can increase the diagnostic yield and enable treatment adaptation based on the specific Ab profile. The detection of causative genetic variants uncovered an underlying hereditary etiology in a significant number of cases, highlighting the importance of distinguishing CMS from autoimmune SNMG due to their contrasting therapeutic approaches.

P49: Antigen discovery in seronegative myasthenia gravis employing a novel in-vitro model of the neuromuscular junction

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Background and aim: Myasthenia gravis (MG) is a rare autoimmune disease of the neuromuscular junction (NMJ) leading to fatigable skeletal muscle weakness. In most patients, autoantibodies against the acetylcholine receptor (AChR, 85 %) or the muscle-specific kinase (MuSK, 5 %) are detectable with commercially available tests such as radioimmunoassay or enzyme-linked immunosorbent assay. The antibody status is important for treatment, as AChR and MuSK patients require different treatments. However, approximately 10 % of patients do not have antibodies against the known targets and are diagnosed as "seronegative MG (SNMG)," complicating diagnosis and treatment in this patient cohort. We hypothesize that SNMG is an antibody-mediated disease caused by low-affinity AChR and MuSK antibodies or yet unidentified pathogenic autoantibodies. Multiple discoveries support the hypothesis that SNMG is caused by antibodies. Immunosuppressive and immunomodulatory therapies have been shown to have a beneficial effect on SNMG and complement depositions have been found at the NMJ of seronegative myasthenia gravis patients. Therefore, we want to improve diagnostics and investigate potential antigenic targets in SNMG.

Method: Using a highly sensitive established live cell-based assay (L-CBA), we assess the prevalence of low-affinity auto-antibodies against MuSK and clustered AChR in SNMG sera. We will use an NMJ model to determine antibody binding to the NMJ proteins. Lastly, we aim to identify novel antigens by immunoprecipitation and mass spectrometry.

Results: To date, we tested over 230 SNMG sera from patients in the D-A-CH region, of which 3.88 % had AChR antibodies. Preliminary results show that 16 out of 159 (10.06 %) SNMG sera display antibodies in proximity of AChR clusters in primary human muscle cells. Furthermore, we have established our mass spectrometry based antigen discovery approach to successfully isolate and identify MuSK and AChR using positive control sera.

Conclusion: We identified anti-AChR antibodies in 3.88 % of seronegative patients due to the lack of sensitivity of commercially available tests. Furthermore, we observe binding of antibodies of SNMG sera close to AChR clusters in a novel in-vitro model of the NMJ, suggesting the presence of unknown antibodies.

P50: Kappa free light chain index and oligoclonal bands in varicella zoster virus infection: A matter of timing?

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Background: Varicella-zoster virus (VZV) remains latent in parts of the nervous system after primary infection and can cause a range of neurological complications. Diagnosis of VZV-related neurological diseases involves clinical findings

and cerebrospinal fluid (CSF) analysis, including the detection of VZV DNA and CSF pleocytosis. However, data on oligoclonal bands (OCB) and κ-free light chain (κ-FLC) index as markers for intrathecal chronic immune reaction are scarce.

Objective: To compare OCB and κ-FLC index positivity in patients with VZV-related neurological diseases.

Method: Patients with the diagnosis of VZV-related neurological disease at the Department of Neurology of the Medical University of Innsbruck were included between 2008 to 2020. OCB were determined by isoelectric focusing followed by immunoblotting and κ-FLC were measured by immunonephelometry. OCB positivity was defined as > 2 cerebrospinal fluid (CSF) restricted OCB, κ-FLC index was considered positive when ≥ 6.1.

Results: A total of 46 patients at a median age of 60 (25th–75th perentile: 45–74) years, comprising 20 (44 %) females, were included in the study. While only 3 (7 %) patients had positive OCB, κ-FLC index (3.61, 2.2–11.8) was elevated in 20 (44 %) patients. While no correlation of κ-FLC index with disease duration was observed (ρ 0.091, p = 0.578), disease duration was longer in OCB positive patients compared to OCB negative patients (21 [15–35] vs. 10 [5–17] days, p = 0.048). The type of disease manifestation (central vs. peripheral nervous system) was not associated with OCB or κ-FLC index positivity.

Conclusion: In VZV-related neurological diseases, positive κ-FLC index was observed more frequently than OCB positivity. κ-FLC synthesis might be also a more sensitive biomarker for acute neuroinflammation, whereas OCB are largely confined to chronic inflammation.

P51: Liquor CXCL13 als ein longitudinaler Biomarker für neurokognitive Beeinträchtigung in HIV-1-assoziierter Demenz nach Therapiestart in HAART-naiven Patient*innen

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Hintergrund: Eine der wichtigen neurologischen Manifestationen der HIV-Infektion ist die HIV-assoziierte Demenz (HAD). Auch mit erfolgreicher antiretroviraler Therapie (ART) mit Suppression der Virusreplikation in Plasma und Liquor finden sich klinische, labortechnische und nuklearmedizinische Hinweise für eine anhaltende Entzündung im ZNS. Diese Entzündung wird bei der HAD im Wesentlichen durch Mikroglia und Makrophagen getragen. CXCL13 ist ein zentrales Chemokin im Zusammenspiel von Makrophagen und B-Zellen und wurde bisher nur in einer Querschnittsstudie bei HAD-Patient*innen im Liquor untersucht.

Fragestellung: Eignet sich die CXCL13-Liquor-Konzentration als Verlaufsmarker für HAD?

Methode: Bestimmung von CXCL13 in longitudinalen Liquorproben bei Patient*innen mit HAD unter neu begonnener ART und Korrelation mit der HAD als klinischer Manifestation und anderen Liquor- und Blutparametern. CXCL13 wurde vor und mehrere Wochen nach Beginn einer ART mittels eines ELISAs (Euroimmun) bestimmt. Das Ausmaß der Demenz wurde mit der Memorial-Sloan-Kettering-Skala bestimmt. Für den Studieneinschluss mussten von allen Patienten die Standard-Liquorwerte, die HIV-Viruslast in Liquor und Plasma, die CD4-Zellzahlen, neuroradiologische und mikrobiologische Befunde vorliegen. Die statistischen Tests waren der Wilcoxon Signed-Rank Test bzw. der Kendall-Rangkorrelationstest.

Ergebnisse: 12 Patient*innen erfüllten die Einschlusskriterien. Bei Baseline war CXCL13 bei 9 Patient*innen nachweisbar, und die Konzentration sank bei allen diesen Patient*innen nach ART-Start. Vor ART-Start bestand eine grenzwertig signifikante Korrelation der Liquorkonzentration des CXCL13 mit dem MSK-Score (τ_b = 0,575, p = 0,051). Im Längsverlauf waren die Minderung der CXCL13-Konzentration und die Besserung des MSK-Scores signifikant positiv miteinander korreliert (τ_b = 0,460, p = 0,048). Weiters korrelierte die Veränderung des Albuminquotienten mit dem MSK-Score (τ_b = 0,714, p = 0,012). Keine Korrelation bestand zwischen dem Verlauf des MSK-Scores unter ART und dem Verlauf der Parameter Liquor- und Plasma-Viruslast und Liquorpleozytose.

Zusammenfassung: Wir untersuchten erstmals den Zusammenhang von Liquor-CXCL13 und HAD im Längsschnitt unter ART. Das Liquor-CXCL13, das von ZNS-ständigen Makrophagen und Mikroglia sezerniert wird, korrelierte dabei besser mit dem klinischen Phänotyp HAD als unmittelbarer mit der HIV-Infektion zusammenhängende Parameter wie Viruslast und Liquorpleozytose. Dies ist vereinbar mit einer bedeutsamen Rolle des CXCL13 in der Pathogenese der HAD und lässt dieses Zytokin als brauchbaren Verlaufsmarker der HAD erscheinen.Zur Validierung unserer Ergebnisse müssten unabhängige Studien mit größeren Teilnehmerzahlen laufen. Deren Rekrutierung dürfte allerdings angesichts des frühen und breiten Einsatzes der ART und der daraus resultierenden Seltenheit der schweren HAD schwierig sein.

P52: Cerebral toxoplasmosis: Pitfalls and spectrum of diagnostics in B-cell lymphoma – A case series

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Introduction: Cerebral toxoplasmosis is a rare encephalitis caused by Toxoplasma gondii, typically occurring in immunocompromised individuals after a period of latent infection. This condition is well documented in patients with HIV or those who have undergone hematopoietic stem cell transplantation, but it is less common in patients with immunocompromising malignancies like lymphoma.

Case series: The first 2 patients, both diagnosed with B-cell lymphoma, developed acute neurological symptoms and new mass lesions in brain imaging. Laboratory tests revealed signs of latent infection with T. gondii and leukopenia in both patients without direct evidence of toxoplasmosis in the cerebrospinal fluid. The first case underwent a brain biopsy with histological confirmation of cerebral toxoplasmosis. They were treated with pyrimethamine and sulfadiazine, resulting in clinical and radiological improvement within a few weeks, therefore confirming the diagnosis of cerebral toxoplasmosis. The third case, a patient with peripheral diffuse large B-cell lymphoma, similarly presented with new neurological deficits and a cerebral mass lesion. Laboratory results showed leukopenia and serological tests revealed positive IgM and IgG for T. gondii. A first brain biopsy was inconclusive. Contrary, this patient did not respond to treatment with pyrimethamine and sulfadiazine within 14 days. A second brain biopsy revealed a cerebral metastasis from diffuse large B-cell lymphoma.

Discussion: This case series highlights the diagnostic uncertainties in cerebral toxoplasmosis for patients with B-cell lymphomas and new cerebral mass lesions. However, given the rare occurrence of brain metastases in patients with peripheral B-cell lymphoma, histologic confirmation should be considered to distinguish cerebral toxoplasmosis. In addition, we provide a comprehensive literature review for cerebral toxoplasmosis in B-cell lymphomas.

P53: The impact of frailty on functional outcome in neurointensive care patients

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Background: Frailty, defined as an age-related syndrome of multidimensional physiological decline, may be more accurate than chronological age alone in identifying brain-injured patients who may benefit from aggressive treatment or, conversely, those in whom unnecessary interventions should be avoided. Here we aimed to evaluate the predictive value of the Clinical Frailty Scale (CFS) score for functional outcome.

Method: This retrospective study included 1008 patients admitted to our neuro ICU between 2018 and 2023. The patients presented with ischemic and hemorrhagic stroke (subarachnoid hemorrhage, SAH and intracerebral hemorrhage, ICH) and traumatic brain injury (TBI). Functional outcome was evaluated with the modified Rankin Scale (mRS) at ICU discharge. Correlations and univariate analyses were used to identify factors associated with higher levels of CFS, and multivariable binary logistic regression analysis was used to assess the potential of CFS to predict poor functional outcome (mRS > 3).

Results: Patients were admitted with an ischemic (n = 256, 25 %) or hemorrhagic stroke (n = 516, 51 %) or TBI (n = 236, 23 %). Patients were 66 (IQR, 54–77) years old and the median length of stay in the ICU was 6 (2–14) days. All severity grades were included, with a median GCS of 14 (9–15) at admission. The median premorbid CFS was 2 (1–3) with 9.2 % classified as frail (CFS ≥ 5) and 76 % of patients taking at least 1 medication . Age correlated with CFS ($r = 0.462$, $p < 0.001$). Patients with chronic illness ($p < 0.001$), prior medication use ($p < 0.001$), and those placed on a "do not resuscitate" (DNR) or "do not escalate" (DNE) regimen ($p < 0.001$) had a higher CFS. In multivariate analysis, adjusted for age, GCS, sex, DNR/DNE status, chronic illness, previous medication use, and disease entity, both a higher CFS and CFS ≥ 5 were independently associated with poor functional outcome (adjOR [95% CI] 1.40 [1.21–

1.62], p < 0.001; 2.83 [1.50–5.33], p = 0.001, respectively). In a subgroup of patients aged ≥ 65 years (n = 532, 53 %), a higher CFS predicted poor functional outcome (adjOR [95% CI] 1.45 [1.20-1.75], p < 0.001). Moreover, a CFS score of ≥ 5 was associated with poor functional outcome (adjOR [95%CI] 2.51 [1.24-5.09], p = 0.011). Age was not associated with poor functional outcome in this subgroup (p = 0.128 and p = 0.095).

Conclusion: In elderly patients, the CFS remained a significant predictor of outcome, while age alone was not associated with poor outcome. This suggests that pre-morbid frailty may provide valuable information for guiding treatment decisions in older neuro intensive care patients, potentially offering more insight than age alone.

P54: Cerebrospinal fluid routine parameters predict functional outcome in spontaneous subarachnoid hemorrhage

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Background: Prognostication in patients with spontaneous subarachnoid hemorrhage (SAH) can be challenging.

Objective: To assess whether cerebrospinal fluid (CSF) red blood cell (RBC) counts and total protein (TP) concentrations, as well as CSF RBC and TP decay rates, are associated with SAH prognosis.

Method: Patients suffering from SAH treated at the neurological intensive care unit of the Medical University of Innsbruck were included in this real-world observational study. Longitudinal CSF samples were collected as part of routine diagnostics. RBC and TP were measured at the time of admission (RBCfirst, TPfirst), in week 1 (RBCDays1–7, TPDays1–7), week 2 (RBCDays8–14, TPDays8–14), or thereafter (RBCDay>14, TPDay>14). The highest detected value (RBChighest, TPhighest), as well as the RBC count adjusted for disease duration (RBCadjusted), were assessed. In patients with a first CSF sample (CSFfirst) within 72 hours after admission and at least 1 subsequent sample, decay rates of RBC and TP were determined. Primary outcome was good functional outcome after 3 months, defined as modified Rankin Scale (mRS) ≤ 2.

Results: A total of 183 SAH patients with a female predominance (69 %), a median age of 60 (50–70) years and median Hunt and Hess score of 4 (IQR 3–5) at admission were included. Multivariable logistic analyses revealed that lower values of RBCfirst, RBCadjusted RBChighest, TPfirst, and TPhighest predicted good functional outcome. Lower TP concentrations during the entire observation time (weeks 1, 2 and 3) and lower absolute RBC measurements early during the disease course (week 1 and 2) were associated with good functional outcome. Similarly, the higher the decay rates of RBC and TP were until week 3, the better the functional outcome was. Reaching RBC and TP below the cut-offs of 1180 cells/µl and 127.5mg/dL, respectively, at week 3 was associated with good functional outcome at month 3.

Conclusion: Lower initial CSF RBC and TP as well as higher CSF clearance predict good functional outcome in SAH patients.

P55: Determinants of brain tissue normoxia in patients with spontaneous intracerebral hemorrhage

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Background: Brain tissue hypoxia (BTH, brain tissue oxygen tension, PbtO2 < 20 mmHg) is common in patients with spontaneous intracerebral hemorrhage (ICH) and contributes to poor outcomes. Research question: Here, we aimed to quantify the prevalence of BTH in ICH patients managed with a PbtO2-guided protocol and to assess determinants of brain tissue normoxia (BTN, PbtO2 ≥ 20 mmHg) and BTH resolution.

Method: In this observational cohort study, we included 58 ICH patients admitted to a neurological intensive care unit between 2010 and 2020 who u▶

derwent multimodal invasive neuromonitoring and were managed with a PbtO2-targeted approach. Brain tissue normoxia was sought by avoiding low cerebral perfusion pressure (CPP) and low blood hemoglobin levels, and by maintaining normocapnia, normoxemia, normothermia, and metabolic homeostasis. One-hour averaged data of continuous PbtO2, CPP, and temperature were matched with intermittent variables (blood gases, hemoglobin, serum glucose, sodium, cerebral microdialysis readings) over a study period of 10 days. Univariate and multivariable regression analyses were performed using generalized estimating equations to account for repeated measurements.

Results: Patients were 61 (IQR, 55–69) years old and presented with an ICH score of 2 (1–3). The overall prevalence of BTH was 31 % and was highest on the first day of monitoring (49 %). In multivariable analysis, the following factors led to the highest prevalence of BTN: CPP 80–89 mmHg (OR 1.88, 95 %CI 1.32–2.68, p < 0.001, reference range: < 60 mmHg), PaO2 90–99 mmHg (OR 1.64, 95 % CI 1.15–2.14, p = 0.001, reference range: < 80 mmHg), central temperature 36.0–37.4°C (OR 2.10, 95 % CI 1.34–3.28, p = 0.001, reference range: < 36.0°C), PaO2/FiO2 100–199 (OR 3.52, 95 % CI 1.60–7.75, p = 0.002, reference range: < 100) in a model corrected for probe position. Out of all potential determinants, only CPP (74.6 [66.8–82.7] vs. 72.5 [64.9-80.2] mmHg, p < 0.001)) was significantly higher after BTH resolution as compared to the time when PbtO2 was lowest during 229 BTH episodes.

Conclusion: BTH is common in ICH patients despite the use of a PbtO2-targeted therapy. Vigilant monitoring and appropriate treatment of determinants, including CPP, are critical in the setting of BTH to achieve BTN.

P56: Post-stroke osteopathy study: Baseline analysis of HR-pQCT parameters and Biomarkers

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Background: Fractures are a frequent problem in post-stroke care leading to a considerable morbidity.[1] Multiple factors lead to this high fracture risk. A systemic bone altering process initiated by the event itself is assumed to play an important role.[2] Considering the poor post-fracture outcome and reduced survival, the prevention of post-stroke fractures is of high importance.[3]

Method: Patients were recruited from the Stroke Card Registry Study (NCT04582825) from March 2021 to May 2023. All patients with ischemic stroke or high-risk TIA (ABCD2-Score ≥ 4) treated at the University Hospital Innsbruck, Austria, were invited to participate in this registry. In a substudy, high-resolution peripheral quantitative computer tomography imaging (HR-pQCT) was performed of the distal radius and tibia right and left site on 4 time points, and blood samples were drawn at 5 time points within 1 year.

Method: 122 patients were included in the study with a mean age of 73.5 years. 27.0 % were women, 13.9 % had a high-risk TIA and 61.5 % were active or former smokers. HR-pQCT parameters and biomarkers at baseline were analyzed.

Results: Differences in nearly all HR-pQCT parameters (except cortical porosity and pore diameter) existed between women and men, with women having a lower total bone mineral density (BMD), as well as reduced BMD in the cortical and trabecular department. There are no differences in HR-pQCT parameters in patients with ischemic stroke and TIA, nor in patients with or without diabetes mellitus, hypertension, and dyslipidemia. Patients who have ever smoked showed a lower total and trabecular BMD, as well as lower values in nearly all trabecular parameters, but no difference in cortical BMD or other cortical parameters. This was true in the same way for active smokers and former smokers. Total BMD decreased with an increasing amount of pack years. No differences in HR-pQCT parameters were found between etiological groups by TOAST.

Biomarker-analyses showed a lower sclerostin in women, but the same levels of osteoprotegerin, sRANKL (soluble receptor activator of nuclear factor- kappaB Ligand), periostin, osteocalcin, and CTX-1 (carboxy-terminal crosslinked telopeptide of type 1 collagen) in women and men. No differences in biomarker levels were found in patients with ischemic stroke and TIA, nor in patients with or without diabetes mellitus, hypertension, dyslipidemia, and non-smokers vs. smokers.

Discussion: In agreement with former

data, women have lower trabecular, cortical, and total BMD.[4] This could be confirmed in this cohort of stroke/TIA patients. The lower total BMD and the significant differences in the trabecular department in active/former smokers vs. non-smokers may reflect the clinical data of Lee et al., where a higher fracture risk after stroke in smoking patients was shown.[5] These differences could not been reproduced in the biomarker analysis. More insights into causes of fractures and bone structure changes (post-stroke osteopathy) after ischemic stroke/TIA are needed to improve fracture prevention.

References:

[1] Batchelor et al., 2012; Ramnemark et al., 2000.
[2] Carda et al., 2009.
[3] Ramnemark et al., 2000.
[4] Whittier et al., 2020.
[5] Lee et al., 2024.

P57: Inzidenz, Charakteristika und Folgen von Frakturen nach akutem ischämischem Schlaganfall oder TIA

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Hintergrund: Post-Stroke Komplikationen treten häufig auf, beeinflussen das funktionelle Outcome und können den Erfolg der Schlaganfall-Akuttherapie mindern. Knochenbrüche sind eine gefürchtete Komplikation, welche die körperliche Beeinträchtigung und Sterblichkeit erhöhen. Frühere Studien berichteten ein bis zu 7-fach erhöhtes Frakturrisiko im Vergleich zur Allgemeinbevölkerung im ersten Jahr nach dem ischämischen Ereignis. Mögliche Ursachen sind neurologische Defizite, ein erhöhtes Sturzrisiko, gemeinsame erworbene Risikofaktoren, genetische Prädispositionen zwischen Fraktur und Schlaganfall, sowie Veränderungen der Knochenstruktur nach einem Schlaganfall („post-stroke Osteopathie").

Zielsetzung: Ziel war es, (a) die Häufigkeit von Frakturen vor und nach einem Schlaganfall/TIA zu bewerten, (b) Prädiktoren für Frakturen zu identifizieren und (c) die Auswirkungen auf Sterblichkeit, funktionelles Outcome und Lebensqualität zu untersuchen.

Methoden: Die Studienpopulation umfasst 2 prospektive Kohorten von Patient*innen mit ischämischem Schlaganfall und TIA, das STROKE-CARD-Register (NCT04582825) und die STROKE-CARD-Studie (NCT02156778) mit ihrem long-term Follow-up (NCT04205006). Patient*innen wurden im Universitätsklinikum Innsbruck, Österreich, rekrutiert. Im prospektiven STROKE-CARD-Register werden alle überlebenden Patient*innen mit ischämischem Schlaganfall oder TIA (ABCD2-Score ≥ 3) eingeladen teilzunehmen. Die Studie umfasst Nachuntersuchungen nach 3 und 12 Monaten. Die randomisierte STROKE-CARD-Studie war ein „open pragmatic intervention trial", das die Auswirkungen eines intensivierten post-stroke Managementprogramms auf das 12-monatige Patient*innen-Outcome und das Rezidivrisiko untersuchte. Bei all diesen Patient*innen wurde zwischen Dezember 2018 und November 2021 ein Langzeit-Follow-up durchgeführt, entweder durch eine Visite in unserer Schlaganfallambulanz oder per Telefonat. Die mediane Nachbeobachtungszeit betrug 5,0 Jahre. Zum Vergleich der Frakturraten zwischen Schlaganfall-/TIA-Patient*innen und der Allgemeinbevölkerung wurde die prospektive Bruneck-Studie herangezogen, eine alters- und geschlechtsstratifizierte Zufallsstichprobe von Einwohnern von Bruneck, Italien.

Ergebnisse: Insgesamt wurden 2.513 Patient*innen eingeschlossen, mit einem medianen Alter von 72 Jahren (IQR 61–79), 39,2 % waren Frauen. Bei 145 Personen (5,8 %, 95%-KI 4,9–6,7) traten 152 Frakturen auf, was einer Inzidenzrate von 61,87 (95%-KI 52,04–71,71) pro 1.000 Personenjahren im ersten Jahr nach dem ischämischen Ereignis entspricht. Es gab keinen Unterschied in den Frakturraten zwischen Schlaganfall- und TIA-Patient*innen (60,84 bzw. 72,28 pro 1.000 Personenjahre). Die Frakturinzidenz war mehr als fünfmal höher als in der Allgemeinbevölkerung (alters- und geschlechtsadjustiertes Hazard Ratio [HR] für die erste Fraktur: 5,36, 95%-KI 2,49–11,52). Das Frakturrisiko war auch 1 Jahr vor dem Schlaganfall/TIA erhöht (HR 2,99, 95%-KI 1,39–6,42). Im Vergleich zwischen Frakturen 1 Jahr vor und 1 Jahr nach dem Ereignis erhöhte Schlaganfall/TIA das Frakturrisiko weiter (alters- und geschlechtsadjustiertes relatives Risiko [RR] 1,69, 95%-KI 1,10–2,58). Die Hauptprädiktoren für Frakturen waren Stürze und Osteoporose. Frakturen nach Schlaganfall/TIA waren ein Prädiktor für Tod (adjustierte Odds Ratio [OR] 2,16, 95%-KI 1,20–3,89), Immobilität (modifizierte Rankin-Skala 4 und 5, adjustiertes OR 2,06, 95%-KI 1,08–3,93) und eine schlechte Lebensqualität.

Zusammenfassung: Patient*innen mit ischämischem Schlaganfall und TIA haben ein hohes Risiko für Frakturen. Frakturen nach Schlaganfall/TIA sind ein starker Prädiktor für Tod, schlechtes funktionelles Outcome und eine reduzierte Lebensqualität. Frakturprävention sollte stärker in die Versorgung nach Schlaganfall oder TIA integriert werden, um das Outcome dieser Patient*innen zu verbessern.

P58: Evaluating a digital health application for post-stroke care and vascular risk factor management

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Background: Stroke is the second leading cause of death among non-communicable diseases and the most frequent cause of permanent disability worldwide. While acute stroke care has significantly advanced over the past 2 decades with the introduction of stroke units, intravenous thrombolysis, and thrombectomy, there remains a scarcity of evidence-based post-stroke disease management programs. STROKE-CARD is one of the few programs with a robust evidence base. As part of the intervention in the randomized controlled STROKE-CARD trial, a simple web-based tool was implemented to provide stroke patients with information and support for vascular risk factor management and healthy lifestyle changes. However, this tool, developed in 2013, highlights a significant gap: More than a decade later, there is still no evidence-based e-health application offering continuous support for stroke survivors. This study aims to evaluate the impact of a newly developed digital health application designed to support ischemic stroke patients after hospital discharge.
Method: The application is designed to empower patients by promoting self-management and providing comprehensive information on prevention, diagnostics, treatment options, stroke rehabilitation, and regional support services. Key features comprise a personalized medication cabinet, appointment reminders for follow-up consultations, and a digital blood pressure diary. The app aims to enhance health literacy and disease management, enabling patients to become more independent and informed. It also addresses tertiary prevention through personalized modules tailored to individual risk profiles, targeting factors such as smoking, obesity, lack of exercise, unhealthy diet, alcohol use, sleep disorders, stress, and depression. Furthermore, the app generates reports to assist healthcare professionals in monitoring blood pressure, physical and mental well-being, vascular risk factors, and in screening for post-stroke complications. The intervention will be evaluated through a multicenter, randomized controlled, open-label medical device investigation with blinded outcome assessment. The study will include 500 patients with stroke or transient ischemic attack (TIA).
Results: As the study is ongoing, results are not yet available. However, the intervention is expected to improve patients' quality of life (EQ-5D), therapy adherence (Medication Adherence Report Scale), health literacy, and stroke risk factor profiles, including blood pressure, body mass index, smoking, alcohol consumption, physical activity (International Physical Activity Questionnaire), nutrition (Mediterranean Eating Pattern for Americans tool), and depression (Hospital Anxiety and Depression Scale). Outcomes will be assessed at 3, 6, and 12 months.
Conclusion: This study will assess the effectiveness of a digital health application in supporting stroke survivors' health management. By enhancing health literacy, therapy adherence, and risk factor management, the intervention has the potential to improve post-stroke care and outcomes. The results will provide valuable insights into the role of digital tools in promoting long-term recovery and tertiary prevention for stroke patients.

P59: Prädiktiver Wert von Serum Neurofilament Light Chain und Glial Fibrillary Acidic Protein für Reperfusionsschäden nach erfolgreicher mechanischer Thrombektomie beim akuten ischämischen Schlaganfall

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Hintergrund: Reperfusionsschäden werden wiederkehrend nach erfolgreicher mechanischer Thrombektomie (MT) eines ischämischen Schlaganfalls beobachtet. Bislang existieren nur wenige Studien zu laborchemischen Biomarkern,

die auf ein erhöhtes Risiko für diese Komplikation hinweisen.
Fragestellung: In unserer Studie untersuchten wir, ob Neurofilament Light Chain (sNfL) und/oder Glial Fibrillary Acidic Protein (sGFAP) im Serum mit dem Auftreten von Reperfusionsschäden und der funktionellen Prognose nach erfolgreicher MT assoziiert sind.
Methode: In dieser prospektiven Beobachtungsstudie wurde sGFAP und sNfL bei Patient*innen mit akutem ischämischem Schlaganfall und einem Großgefäßverschluss in der vorderen Zirkulation mithilfe des Simoa 2-plex Advantage Assay (Simoa HD-X Analyzer) innerhalb von 6 Stunden nach einer erfolgreichen MT (TICI 2b-3) analysiert. Alle eingeschlossenen Patient*innen wurden 24 bis 96 Stunden nach MT mittels Magnetresonanztomografie des Gehirns untersucht. Reperfusionsschäden wurden definiert als das Vorhandensein von vasogenem Ödem und/oder hämorrhagischer Transformation (HT) des Infarktgebiets. Die Bewertung des funktionellen Outcome 3 Monate nach dem Schlaganfall erfolgte anhand der modifizierten Rankin-Skala (mRS). Ein schlechtes klinisches Outcome wurde als mRS 3–6 definiert.
Ergebnisse: Von 127 Patient*innen (medianes Alter: 74 Jahre; 59,1 % weiblich) wurde bei 60 (47,2 %) ein Reperfusionsschaden identifiziert (vasogenes Ödem: n = 48; HT: n = 38). sGFAP (Median: 2.098,6 pg/ml, Interquartilsabstand [IQA]: 698,5–6.978,5) zeigte eine unabhängige Assoziation mit Reperfusionsschäden (adjustierte Odds Ratio [aOR]: 3,5; 95%-Konfidenzintervall [KI]: 1,6–7,7; p = 0,002) sowie mit einem schlechten klinischen Outcome 3 Monate nach dem Schlaganfall (aOR: 3,0; 95%-KI: 1,3–6,7; p = 0,009). Für sNfL konnten hingegen keine derartigen Zusammenhänge festgestellt werden (Median: 34,6 pg/ml, IQA: 21,2–50,8; p > 0,1).
Konklusion: sGFAP könnte Patient*innen mit einem erhöhten Risiko für Reperfusionsschäden nach einer erfolgreichen MT anzeigen, während sNfL in diesem Zusammenhang keinen prädiktiven Wert aufweist. Eine Biomarker-basierte Risikostratifizierung könnte individuelle Präventionsstrategien fördern.

P60: Cerebral infarction due to an electrical accident: A case report

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Background: The extent of the damage to the organism depends on the type and strength of the current, the age and constitution of the patient, the clothing, and the duration of the exposure. The electrical resistance in particular is not constant, but depends on many factors, e.g., dry/wet ground, protective clothing (shoes with thick soles, etc.), and weather conditions (thunderstorms).
Research question: Cerebral vasospasm due to electrical accident?
Case: A 38-year-old man was admitted to the emergency room after a 400V electrical accident. The neurological examination revealed incomplete left oculomotor nerve paresis and dizziness with an unsteady gait. No abnormalities were found in the acute cCT including CTA. The acute MRI also showed only nonspecific hyperintense white matter spots on both hemispheres, but no evidence of recent ischemia. Over time, the patient became somnolent. A subsequent cMRI revealed a new bilateral diffusion restriction in the thalamus and a diffusion restriction on the left paramedian cerebral crus, which is consistent with an ischemic infarction with atypical supply (Percheron's artery). An echocardiogram found a persistent foramen ovale. This suggests that a paradoxical embolism or cerebral vasospasm due to the electrical accident is a possible cause.
Results: At the time of discharge, the occulomotor nerve palsy was gradually recovering and the neuropsychological evaluation showed mild deficits in shared and selective attention and verbal memory.
Summary: The possible pathomechanism in the patient described above is that cerebral vasospasm led to occlusion of the Percheron's artery. Alternatively, a paradoxical embolic event due to a persistent foramen ovale is a possibility

P61: Hereditary spastic paraplegias in Austria

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Background: Hereditary spastic paraplegias (HSPs) are rare inherited neurodegenerative disorders. Clinically, they can be divided into pure (pHSP) and complicated (cHSP) forms. While all share the core clinical features of weakness and spasticity of the lower limbs, cHSP can exhibit additional signs and symptoms, such as ataxia, cognitive impairment, or neuropathy. To date, more than 80 distinct mutations and genes associated with HSP have been described. The estimated prevalence of HSPs is 3–10/100000.

Objective: We report the data of the HSP patient cohort from the Center for Rare Neurological Diseases in Innsbruck.

Method: Retrospective data from all patients with a clinical diagnosis of HSP at our center were included in the analysis. Furthermore, a prospective registry was initiated and all subjects included between April 2019 and August 2024 were analyzed. The Spastic Paraplegia Rating Scale (SPRS) and Scale for the Rating and Assessment of Ataxia (SARA) were used to assess disease severity.

Results: A total of 129 patients, 84 (65.1 %) of whom were male, were included in the study. Sixty-four patients (50.4 %) had pHSP. The mean age at visit was 44.1 (SD 17.7) and the mean disease duration 16.5 (SD 11.7) years. Genetic diagnosis was confirmed in 56.6 % of the cases. The most common genotype was SPG4 (23.3 %), followed by SPG11 (7.8 %) and SPG7 (4.7 %).

Eighty subjects, 54 male (67.5 %) and 26 female (32.5 %), were included in the prospective registry study. Again, SPG4 (26.3 %) was the predominant genotype, followed by SPG11 (12.5 %) and SPG7 (6.3 %). Twenty-nine patients (36.3 %) underwent only baseline evaluation, while 5 patients (6.3 %) underwent 5 follow-ups. A total of 216 visits were conducted. Forty-one (51.2 %) patients had pHSP. The mean SPRS score was 18.2 (SD 10.7) and the mean SARA score 7.8 (SD 7.5).

Conclusion: We analyzed the datasets of 129 patients, 80 of whom were included in this prospective registry study. In accordance with other registry studies, SPG4 is the predominant genotype in our cohort. Disease severity, as measured by the SPRS, was similar to that in a larger German cohort. The rate of pHSP in our cohort, however, was higher than in other studies. Additionally, male predominance was more marked in our cohort than in French and German studies. These results are limited by the fact that this was a single-center study.

In conclusion, this is the largest cohort of patients in Austria published to date and provides important insights into the frequency and clinical spectrum of this rare disease in Austria. As this is an ongoing project with active recruitment, we would like to invite all our colleagues to refer patients with HSP to our site in order to be able to depict a more complete picture of HSP in Austria.

P62: Resting-State EEG analysis defines the signature of CACNA1A and GAA-FGF14 related channelopathies

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Background: The CACNA1A gene encodes the pore-forming subunit of P/Q voltage-gated calcium channels, crucial for presynaptic neurotransmitter release. CACNA1A variants are associated with neurological phenotypes sharing a common denominator of chronic cerebellar signs and paroxysmal features, such as spells of episodic ataxia. A clinically overlapping phenotype has recently been linked to a pathologically expanded intronic GAA-repeat in the FGF14 gene encoding fibroblast growth factor which regulates the function of both potassium and voltage-gated sodium channels.

Research question: Channelopathies have been associated with a variety of resting-state electroencephalogram (EEG) alteration. Sparse evidence suggests that CACNA1A variants alter EEG patterns, though these effects are not well-quantified. Moreover, data on EEG patterns in the phenotypically related GAA-FGF14 ataxia are lacking.

Method: This study examined differences in relative EEG band powers and functional connectivity among patients with CACNA1A variants, GAA-FGF14 ataxia, and matched healthy controls (HC) randomly selected from a public dataset. Resting-state EEGs in CACNA1A and GAA-FGF14 ataxia patients were recorded in the EEG laboratory of the Department of Neurology of the Medical University of Innsbruck using the 10-20 system. Signals were bandpass filtered (0.5–40 Hz), segmented into 2-second epochs, and electrooculographic artifacts were removed automatically using inde-

pendent component analysis. Relative bandpower was calculated using Welch's method, and functional connectivity was assessed through the weighted Phase-Lag Index (wPLI) and Minimum Spanning Tree (MST) metrics. Bayesian linear regression evaluated genotype effects on EEG metrics, with significance defined by the 95 % highest density interval (HDI95%). Age and sex differences between the 2 patients' groups necessitated including these as covariates. To enhance statistical power, the relationships between covariates and EEG metrics were estimated using an independent HC cohort, and posterior distributions from this analysis informed priors for group comparisons.

Results: A total of 82 subjects were analyzed, including 29 CACNA1A patients, 15 GAA-FGF14 ataxia patients, and 30 HC. Overall relative theta power was increased in CACNA1A patients compared to both GAA-FGF14 patients (mean difference [MD]: 0.027, HDI95%: [0.005,0.049]) and healthy controls (MD: 0.037, HDI95 %: [0.008,0.066]). FGF14 patients consistently exhibited higher relative beta and relative gamma power than CACNA1A patients (parietal: MD: 0.014, HDI95%: [0.004,0.025]) and HC. CACNA1A patients showed isolated decreased relative delta power in frontal channels compared to HC (MD: 0.09, HDI95%: [0.015,0.169]). Functional connectivity analysis revealed significantly increased overall wPLI in the theta and delta bands in CACNA1A patients compared to HC (theta: MD: 0.061, HDI95%: [0.009, 0.115]). Additionally, MST maximum degree centrality in the delta band was increased in CACNA1A patients compared to controls (MD: 0.059, HDI95%: [0.014, 0.106]) indicating higher degree of centralization of the connectivity graph.

Conclusion: The present preliminary findings demonstrate distinct resting-state EEG alterations in CACNA1A patients compared to the phenotypically related GAA-FGF14 disease and to HC, highlighting the unique network-level consequences of P/Q-type calcium channel dysfunction. This study underscores the potential of advanced EEG analysis as disease state read-out in calcium channelopathies.

P63: Clinical clues for a genetic etiology in late onset cerebellar ataxia

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Background: Late-onset cerebellar ataxia is defined as sporadic onset of ataxia after the age of 40. Their etiology ranges from acquired to genetic causes. Until recently, up to 80–90 % of late-onset ataxias remained unexplained, leading to a diagnosis of idiopathic late-onset cerebellar ataxia (ILOCA). Recently, 2 new causes for late-onset cerebellar ataxia were identified, namely the intronic repeat expansion in the FGF14 gene causing spinocerebellar ataxia type 27b (SCA27b) and the intronic repeat expansions in the RFC1 gene causing CANVAS.

Aim: This retrospective observational study aimed at assessing distinct clinical presentation of late-onset cerebellar ataxias in the light of newly discovered genes.

Method: Patients presenting to the ataxia outpatient clinic at the Center for Rare Movement Disorders Innsbruck (CRMDI) at the Medical University of Innsbruck between 1998 and 2024 with onset of disease after age 40, with a negative family history, and non-fulfilling the criteria of a probable MSA-C according to the Gilman criteria were included. Annual clinical follow-ups and adapted genetic evaluation was performed in this cohort.

Results: In our cohort, 30 patients (18 male, 12 female) were identified as ILOCA. Among these, stepwise molecular testing during follow-up identified 15 cases with SCA27b, 2 with CANVAS, and 5 with SCA6. No genetic cause was found in 8 patients. Dysphagia was reported in 4 (= 50 %) of ILOCA patients but none of the patients with genetic diagnosis ($p < 0.001$). Episodic symptoms including intermittent gait disturbance, blurred vision, or vertigo were reported by 16 out of 22 patients (77.3 %) with genetically assigned ataxia ($p < 0.001$). The most common initial episodic symptom was episodic gait ataxia, which occurred in 3 (60.0 %) of SCA6 and 8 (53.3 %) of SCA27b patients but in none of the CANVAS or ILOCA patients. Vertical nystagmus was present in 13 patients (59.1 %) with genetically assigned ataxia, but only one (12.5 %) ILOCA patient, while horizontal nystagmus was present in 81.4 % and 62.5 %, respectively.

Discussion: Our study highlights the importance of recognizing episodic symptoms such as gait disturbance, vertigo, or double vision as potential indicators for a genetic etiology of a late-onset cerebellar ataxia. Clinicians should prioritize genetic testing in patients presenting with episodic symptoms to streamline the diagnostic process. Targeted genetic testing not only improves diagnostic accuracy but also reduces unnecessary diagnostic procedures, lowering the financial burden on healthcare systems. Furthermore, episodic fluctuations may markedly contribute to disease burden and are potentially amenable to pharmacological treatment.

P64: Spinocerebellar ataxia type 2 in Austria: A thirty-year, multigenerational follow-up

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Background: Spinocerebellar ataxia type 2 (SCA2) is an autosomal dominant cerebellar ataxia caused by a CAG triplet repeat expansion in the Ataxin 2 gene, characterized by gait ataxia and brainstem oculomotor dysfunction. The disease-causing mutation was first identified in a Cuban cohort in 1993.
Aim: The goal of this prospective observational study was to observe the natural history of SCA2 to increase disease knowledge and shorten time to diagnosis in affected individuals.
Method: Over a time period of thirty years (1994–2024), 7 families of patients with symptomatic SCA2, including 35 individuals as well as 7 SCA2 patients not related to any of those families, were regularly examined by a team from the Center for Rare Movement Disorders Innsbruck. To minimize reporting bias regarding disease onset, both genetically confirmed patients as well as clinically unaffected individuals from later generations were examined. Disease onset was defined as the reported onset of gait difficulties in patients who were symptomatic at first presentation and a score > 3 points on the Scale for the Assessment and Rating of Ataxia (SARA) in first-degree relatives.
Results: A total of 42 individuals (17 M, 25 F) were included in the study. Of 9 first-degree relatives clinically not affected at the time of first presentation, 3 had developed ataxia by the end of 2024. The average repeat expansion increase of the disease allele across generations was 2 (range -2–6) CAG repeats. The corresponding average anticipation of disease onset was 8.7 years (SD 13.8). The median onset of disease in patients with less than 38 repeats (n = 5) was 40 years (25th–75th percentile 39–51), while patients with 38 to 42 repeats (n = 21) had a median onset of disease at 35 years (40–44). Patients with more than 43 repeats (n = 10) had a median onset of disease at 21 years (20–27.5). Cramps as an early symptom of motor neuron involvement were present in 73.5 % overall, ranging from 60.0 % of patients with less than 38 repeats to 90.0 % in patients with more than 44 repeats. Amyotrophy as a late symptom of motoneuron involvement was present in 20.0 % of patients with < 38 repeats, 57.9 % of patients with 38–42 repeats, and 90.0 % of patients with > 42 repeats, respectively.
Discussion: Our study highlights the importance of long-term, multigenerational follow-ups for the understanding of the natural history of rare diseases. By including individuals based on their family history without a genetic confirmation of a hereditary disease, a more accurate picture of disease onset may be made. Accurate, complete, and comprehensible patient information is a crucial point in the ethically correct conduct of such studies.

P65: Types of pain in multiple system atrophy

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Background: A recent web-based survey showed that 87 % of individuals with multiple system atrophy (MSA) report pain. However, it remains unclear which types of pain mostly contribute to pain burden in this rare disease.
Research question: We aimed to estimate the prevalence of different pain types in MSA by means of the King's Parkinson's Disease Pain Questionnaire (KPPQ) and of structured questions addressing putative MSA-specific pain types, and to compare this with a historical cohort of age-, gender-, and disease duration-matched individuals

with Parkinson's disease (PD) and healthy controls (HCs).

Method: We first analyzed the prevalence of different pain types according to the KPPQ (distinguishing between musculoskeletal, chronic, fluctuation-related, nocturnal, orofacial pain, pain related to discoloration, oedema/swelling, and shooting pain/pins and needles) in a community-based cohort of MSA individuals, who answered a web-based survey in 2023. We additionally assessed the prevalence of further putative MSA-specific pain types (i.e., coat-hanger pain, pain related to catheterization, recurrent urinary infections, bladder spasms, pressure sores, bruises, and painful cold hands and feet). For comparison, MSA individuals were afterwards matched for gender, age (± 3 years), and disease duration (± 2 years) with PD subjects and HCs from the King's College historical cohort, who had completed the KPPQ.

Results: 264 MSA individuals accessed our survey. After data cleaning, 194 questionnaires were retained for final analysis, 157 with completed KPPQ. Individuals suffering from MSA reported more frequent nocturnal (73 %, n = 114), musculoskeletal (63 %, n = 98), and fluctuation-related pain (62 %, n = 94). Among other putative MSA-specific pain types, coat-hanger pain (59 %, n = 91), painful cold hands and cold feet (48 %, n = 75), and pain related to bruises (44 %, n = 69) were most frequently reported. No differences in the prevalence of any type of pain were observed across the parkinsonian (MSA-P, n = 59) and the cerebellar (MSA-C, n = 75) subtype. Ninety-six individuals with MSA were matched for age, gender, and disease duration with persons with PD and HCs. All pain types were more frequent in MSA compared to HCs except for musculoskeletal pain, which occurred as frequently in MSA as in the HCs (63 % vs. 66 %, p = 0.722). Conversely, musculoskeletal pain was more common in PD compared to MSA (78 % vs. 63 %, p = 0.023), while orofacial pain was more frequently reported by persons with MSA compared to PD (32 % vs. 12 %, p < 0.001).

Conclusion: In MSA, both disease-related (e.g., coat-hanger pain due to orthostatic hypotension) and -unrelated (e.g., musculoskeletal pain) types of pain contribute to pain burden. While pain assessment tools not specifically designed for MSA may help to catch disease-unrelated types of pain, tailored tools may be needed to identify disease-specific pains in MSA, which may benefit from an optimized symptomatic management of MSA core motor and non-motor features.

P66: Optic coherence tomography: A possible biomarker in early Huntington's disease.

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Background: Huntington's disease (HD) is a neurodegenerative disorder characterized by motor, cognitive, and behavioral abnormalities. As therapeutic trials are ongoing, there is an urgent need for accurate biomarkers to monitor neurodegeneration. Optical Coherence Tomography (OCT) offers a non-invasive method to measure retinal changes that reflect neurodegenerative processes, potentially also in HD.

Objective: To assess the role of spectral domain OCT as a biomarker in Huntington's disease.

Method: This cross-sectional study compared spectral domain OCT data in HD patients and healthy controls (HC). HD patients were classified into stage1 and stage 2 based on motor symptoms and functional capacity.

Results: We recruited a total of 68 participants, including 39 HD patients (22 stage 1, 17 stage 2) and 29 age-matched HC. There were no significant differences in age and gender between the groups. Stage 2 HD patients showed worse motor function (UHDRS-TMS 28.44 ± 18.13 vs. 13.74 ± 8.78, p = 0.002) and functional capacity (UHDRS-TFC 8.13 ± 2.03 vs. 12.44 ± 0.99, p < 0.001) and lower scores on the MMSE (27.36 ± 1.64 vs. 28.73 ± 1.74, p = 0.005 vs. 29.45 ± 0.91, p < 0.001) compared to stage1 HD patients and HC respectively. Both stage 1 and stage 2 HD groups displayed significantly reduced macular retinal nerve fibre layer thickness (mRNFL) (33.45 ± 4.70, 31.90 ± 3.47 vs. 38.45 ± 5.00; p < 0.01) and ganglion cell-inner plexiform layer thickness (GCIPL) (71.63 ± 6.38, 60.42 ± 4.67 vs. 77.03 ± 8.40, p < 0.01) as compared to HC. However, no significant OCT differences were found between stage 1 and stage 2 HD stages. The retinal OCT parameters mRNFL and GCIPL correlated moderately with PINHD (r = -0.424, r = -0.513 with ▶

all p-values < 0.001) and CAP (r = -0.425, r = -0.482; all p-values < 0.001). Further, GCIPL showed moderate correlation with MMSE scores (r = 0.436 with all p-values < 0.001).

Conclusion: HD patients exhibit significantly thinner retinal ganglia cell and macular retinal nerve fibre layer compared to healthy controls, even in early disease stages. These findings suggest that OCT may serve as a valuable biomarker, potentially suitable to track disease progression, especially in the early stages of HD.

P67: Genome aggregation database version 4: Allele frequency changes and impact on variant interpretation in dystonia

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Background: Population-scale databases are central to the assessment of variant-allele frequencies. Although the recently released Genome Aggregation Database (gnomAD) v4 offers a more than fivefold increased sample size compared to v2, the implications of the additional data on variant analysis in dystonia are unknown.

Objective: To evaluate population-frequency changes of variants associated with monogenic dystonia.

Method: All curated variants linked to the 6 most common dominant forms of dystonia were extracted from the MDSGene catalogue. Their population frequencies were compared between gnomAD v2.1.1 and v4.0. Constraint metrics for each gene were analyzed.

Results: The majority of dystonia-associated variants (192/247, 77.7 %) remained absent from both gnomAD versions. Most of the 28 variants already documented in v2.1.1 showed no relevant allele-frequency changes. Of 219 variants not listed in v2.1.1, 27 (12.3 %) appeared for the first time in v4.0. Six of these variants had an absolute count of ≥ 4, including well-established pathogenic alleles. Gene constraints were unaltered, except for GNAL and KMT2B.

Conclusion: MDSGene-registered variants in dystonia are mostly ultra-rare, but a growing number of disease-linked alleles is seen in the populations represented by gnomAD. Presence of a variant observed in a patient with dystonia in v4 data does not preclude its pathogenicity.

P68: Gender-Aspekte bei der Huntington-Krankheit: Eine retrospektive Analyse

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Hintergrund: Die Huntington-Krankheit ist eine neurodegenerative Erkrankung, die durch fortschreitende motorische, kognitive und psychische Symptome gekennzeichnet ist. Die Datenlage zu geschlechtsspezifischen Unterschieden hinsichtlich Krankheitsverlauf ist uneinheitlich und betont die Notwendigkeit weiterer Untersuchungen zur Entwicklung gezielter Behandlungsstrategien.

Fragestellung: Gibt es geschlechtsspezifische Unterschiede hinsichtlich des Alters bei Beginn verschiedener Krankheitssymptome?

Methodik: Es wurde eine retrospektive Analyse von Huntington-Patient*innen der Chorea-Ambulanz der Universitätsklinik für Neurologie Innsbruck durchgeführt. Dabei wurden die Krankheitssymptome in motorische und nichtmotorische Symptome eingeteilt und das Alter zu Beginn der jeweiligen Symptome geschlechtsspezifisch untersucht. Die Auswertung erfolgte unter Berücksichtigung der CAG-Wiederholungszahl.

Ergebnisse: Insgesamt wurden die Daten von 72 Huntington-Patient*innen analysiert, davon waren 40 weiblich und 32 männlich. Es fanden sich keine signifikanten geschlechtsspezifischen Un-

terschiede hinsichtlich der CAG-Wiederholungen (p = 0,298) sowie hinsichtlich des Alters zu Beginn der motorischen bzw. nichtmotorischen Symptome.

Zusammenfassung: In dieser retrospektiven Auswertung zeigte sich kein geschlechtsspezifischer Unterschied im Alter beim Auftreten motorischer oder nichtmotorischer Symptome. Große Registerauswertungen deuten jedoch daraufhin, dass Frauen eine schnellere Krankheitsprogression haben könnten als Männer und das Geschlecht einen komplexen Einfluss auf das phänotypische Erscheinungsbild und den Krankheitsverlauf der Huntington-Krankheit hat. Diese Erkenntnisse betonen die Notwendigkeit weiterer Forschung, um geschlechtsspezifische Unterschiede besser zu verstehen und gezielte Therapieansätze zu entwickeln.

P69: Parkinson's disease prodromal and risk factors in a European population-based cohort: The Healthy Brain Aging (HeBA) study

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Background: The MDS prodromal PD criteria integrate several prodromal and risk factors for PD including positive family history, REM sleep behavior disorder, and olfactory dysfunction.[1] Smell tests, such as the University of Pennsylvania Smell Identification Test (UPSIT) have been commonly used in PD risk screening studies. Here, we report results of a large European online survey assessing PD-associated risk factors.

Objective: To describe the frequency of prodromal and risk factors for Parkinson's disease (PD) in a large European population-based cohort and to analyze the predictive value of online survey single questions in detecting hyposmia as a risk marker for PD.

Method: The Healthy Brain Aging (HeBA) study used an online questionnaire targeting PD risk and prodromal factors, including high-interest single questions (HIQ) on smell loss, dream enactment behavior, family history of PD, and subjective cognitive decline alongside validated scales. A subset of individuals, the majority of whom were considered to be at high-risk of prodromal PD, underwent remote olfactory assessment using the UPSIT,[2] with hyposmia defined as age- and sex-adjusted percentile below 10. A subset of participants was invited for in-person clinical evaluation. Data collection occurred at 4 European sites (Kassel, Innsbruck, Luxembourg, and Barcelona), together forming the HeBA-cohort.

Results: The HeBA-cohort includes 29430 individuals (59.4 % women, mean age 62.9 [± 7.8] years). Most participants (78 %) were highly educated and independent (99 %). Subjective smell loss was reported by 8.3 % of participants, 14.7 % endorsed symptoms compatible with dream enactment behavior, 18.3 % had a positive family history of PD, and 12.9 % had a first-degree relative with PD. In addition, 19.6 % experienced subjective cognitive deficits. Of all participants, 4.5 % answered positively to 2 HIQ, and 0.4 % to 3. No significant site differences were observed among the distribution of positive HIQ by age. Among UPSIT completers (n = 8428), 61.4 % were women, mean age 62.9 years. Subjective smell impairment was reported by 16.5 %, which was associated with abnormal UPSIT percentiles. Sensitivity for hyposmia was low at 34.5 %, with 87.6 % specificity.

Conclusion: The HeBA study es- ▶

tablished a well-characterized cohort for exploring PD risk and prodromal factors in the healthy European population. Remote olfactory function assessment has been deemed feasible and could potentially be used as an early PD screening tool. Longitudinal follow-up and inclusion of biological markers will enhance understanding of early PD phases.

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P70: A biallelic repeat expansion mutation in RFC1 as a risk factor for Parkinson's Disease

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Introduction: Repeat expansion mutations (REMs) residing in the replication factor complex unit 1 (RFC1) have recently been implicated in the pathogenesis of Parkinson's disease (PD). The biallelic RFC1 expansion **[**AAGGG**]**_n has first been identified in association with the neurological disorder CANVAS.

Aim: Our main aim was to screen a large cohort of PD patients for REM variants of RFC1 to evaluate whether their frequency differs from neurologically healthy controls. Additionally, we assessed the question of whether PD patients carrying RFC1 REMs clinically differed from typical PD or CANVAS patients.

Method: We applied repeat primed PCR, Southern blot analyses and flanking PCR on a large cohort of Austrian patients with clinically diagnosed parkinsonism to screen for different RFC1 expansion variants.

Results: X1.34 % of our PD patient cohort (12/1204) were found to carry biallelic **[**AAGGG**]**_n expansions compared to 0.7 % of healthy controls (1/1204). This strongly emphasizes the relevance of this RFC1 genotype in the etiology of PD. Interestingly, we also observed a correlation of the biallelically expanded wildtype RFC1 motif **[**AAGGG**]**_11 with the development of PD, which was so far reported to not result in pathogenic phenotypes. Southern blot analysis of biallelic **[**AAGGG**]**_n expanded PD samples showed smaller expansions, with the larger allele containing 380–660 repeat units compared to CANVAS patients, which typically range from 400–2000. All samples also had one allele close to wild-type size. We postulate that individuals with 1 near-wild-type RFC1 expansion and a second expansion above the pathological threshold but below CANVAS levels may be more likely to develop PD rather than CANVAS. Clinical assessment of PD patients with the biallelic **[**AAGGG**]**_n expanded genotype showed that 87.5 % lacked a clear levodopa response, despite its importance as a PD diagnostic criterion. None of those patients exhibited CANVAS-like features.

Conclusion: Our study shows a strong association between biallelic RFC1 repeat expansions and the development of Parkinson's disease. These expansions are smaller than those seen in CANVAS but exceed normal repeat sizes. Consequently, our findings yield great importance regarding future genetical diagnostic in clinical context.

P71: Genotype and age at onset drive vermis atrophy in CACNA1A and GAA-FGF14 related ataxias

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Background: Ion channel dysfunction is a recurring etiology in the heterogenous genetic landscape of inherited ataxias. Ataxic syndromes associated to channelopathies typically present with both chronic and episodic neurological symptoms and usually show an exclusive or prevalent involvement of the vermis in imaging studies. These features are recapitulated by CACNA1A (MIM 601011) and FGF14 (MIM 601515) related disorders, which list among the most frequent molecular etiologies of

inherited ataxias

Research question: In the present study, we applied a deep learning method for the lobular segmentation of the cerebellum to assess cerebellar volumetry in a cohort of patients with cerebellar ataxia due to either non-polyglutamine CACNA1A variants or GAA-FGF14 related disease. Our aims were i) to evaluate the diagnostic performance and discriminative power of this novel methodology and ii) to describe possible distinct molecular and clinical correlates based on neuroanatomical mapping-based discrimination.

Method: MRI acquisitions were performed on a 3.0 Tesla whole-body Siemens MR scanner. 3-dimensional T1-weighted images were processed with the FastSurfer pipeline and the CerebNet module to segment subcortical brain region and estimate different brain regions' volumes. Normative data for subcortical regional volumes over the lifetime of the adult human brain were generated from healthy participants from open-access MR datasets. After z-transformation, a k-means clustering analysis with 3 centers was performed to group the data points into distinct clusters based on their similarity, aiming to identify underlying patterns of infratentorial brain atrophy. Subsequently, a principal components analysis (PCA) of the cluster dimensions was conducted to study what dimensions primarily drive cluster separation.

Results: In the cluster analysis, cluster 1 contains 6 patients carrying missense CACNA1A variants who had an early onset of disease (age 7, range 1–12 years) and present with hemiplegic migraine as an episodic feature ($p = 0.004$). Patients in cluster 2 ($n = 16$), and cluster 3 ($n = 6$) presented mixed genotypes, but an increasing frequency of episodic ataxia as paroxysmal manifestation ($p = 0.028$) and an increasing age at onset ($p = 0.01$) were observed from cluster 1 to cluster 2. The PCA revealed that 2 principal components account for 83 % of the total variance in the dataset, with the first principal component explaining 78 % of the variance, indicating that it captures the most significant patterns. Vermis atrophy as well as the cerebellar lobules IV–VII had the highest relative contribution to this principal component. Cerebellar volume loss did not correlate with the age at the examination nor with the severity of the chronic cerebellar syndrome as expressed by clinical scores.

Conclusion: Our study suggests that the age at onset and the molecular mechanism of channel dysfunction are the most important determinant of cerebellar volume loss in CACNA1A and GAA-FGF14 disease.

P72: Novel diagnostic for multiple system atrophy: Combining AI and decision analysis

Kuchin I[1], Jahn B[1], Sroczynski G[1], Santamaria J[1], Schmid D[1, 2], Arvandi M[1], Krismer F[3], Schrag A[4], Schwingenschuh P[5], Fanciulli A[3], Siebert U[1, 6, 7]

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Background: Multiple system atrophy (MSA) is a rare, fatal, and rapidly progressive neurodegenerative disease. Differentiating between MSA and Parkinson's disease (PD) is challenging, and MSA may be misdiagnosed. This study aims to support the development of an AI-based diagnostic tool that incorporates magnetic resonance imaging (MRI) evaluations of the brain and provides predictive probabilities the presence of MSA and PD. In this case, medical decision making must find the optimal probability threshold (i.e., cutoff) based on the different sensitivity and specificity values along the receiver-operating characteristic (ROC) curve accounting for long-term consequences of the diagnostic decision, that is, the benefits and harms from true/false positive/negative test results.

Research question: What is the comparative benefit-harm balance of different cutpoints on the ROC curve of AI-based imaging diagnostics for patients with MSA?

Method: To optimize the AI-based MSA diagnostic tool currently investigated by the Medical Decision Making in MSA Research Group (MeDeMSA, www.i-med.ac.at/medemsa), a decision-analytic optimization framework was developed. The decision population includes patients presenting with parkinsonian symptoms at specialized clinics. The primary outcome is quality-adjusted life expectancy (QALE) expressed in quality-adjusted life years (QALYs), which synthesizes the benefits and harms assessed over patients' remaining lifetimes. Using a probability-based decision tree model to outline and systematically quantify the outcomes for correctly classified patients with MSA and PD and those who were misclassified, we identified cutpoints along the ROC curve. In the initial stage of the framework development, the model was informed by published ▶

ROC curve data. Model parameters include an MSA prevalence of 12.5 % from the Innsbruck Movement Disorder Clinic. To calculate QALE, we used an average quality-of-life index (so called "utility") of 0.73 at the time of diagnosis, and an updated diagnosis after 5 years due to disease progression. Utility decrements over time were based on cohort studies and expert opinions, incorporating the impact of disease severity and progression and treatment and care effects.

Results: Our decision tree model showed that the overall QALE was 8.75 QALYs for a cutoff with sensitivity = 0.001 and specificity = 0.999) and 8.67 QALYs at sensitivity = 0.999/specificity = 0.001. The optimal cutpoint could be achieved with the current diagnostic with sensitivity = 0.621 and specificity = 0.953, leading to an overall QALE of 8.76 QALYs. Results are particularly sensitive to the assumed prevalence of MSA and potential effectiveness of disease-modifying treatment for patients with MSA. Higher prevalence and the availability of a disease-modifying treatment favor cutpoints with higher sensitivity.

Summary: Our study showed that the decision-analytic framework can be applied to optimize novel AI-based MSA diagnostic tools accounting for consequences for the diagnostic decision and benefit-harm long-term consequences. In the next step, AI-based diagnostic results from the MeDeMSA study group will be incorporated into the model as soon as they become available. In addition, a Delphi-survey on potential effects of disease-modifying treatment on life expectancy and quality of life of MSA patients is under development to inform potential future diagnostic consequences and, in the end, medical decision making.

P73: Evaluating personalized best medical care for MSA: A decision-analytic framework

Kuchin I[1], Jahn B[1], Sroczynski G[1], Santamaria J[1], Schmid D[1, 2], Arvandi M[1], Krismer F[3], Schrag A[4], Schwingenschuh P[5], Fanciulli A[3], Siebert U[1, 6, 7]

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Background: Multiple system atrophy (MSA) is a rare neurodegenerative movement disorder for which no effective treatment exists. Personalized care protocols tailored to individual patient needs can mitigate the decline in quality of life (QoL). The Medical Decision Making in MSA Research Group (MeDeMSA, www.i-med.ac.at/medemsa) has initiated a clinical trial to evaluate personalized best medical care (PBMC) with or without telemedicine (TM). This project aims to develop, validate, and apply a decision-analytic model based on data from the trial and additional long-term evidence to assess long-term effectiveness, cost-effectiveness, and cost-utility of PBMC and TM compared to usual care in patients with MSA to inform decision makers.

Research question: What are the structure, main components, model input data sources and simulation approaches of a decision-analytic framework for evaluating the long-term comparative outcomes of PBMC with or without TM compared to usual care in patients with MSA?

Method: A decision-analytic framework was developed to guide future model design and software implementation. The framework was informed by a literature review and input from an interdisciplinary international expert panel comprising medical professionals, quality of life specialists, end of life care experts, epidemiologists, ethicists, health technology assessment (HTA) specialists, and decision science experts. The panel convened for hybrid meetings and follow-up discussions. The framework includes the research question, target population, compared interventions, time horizon, health states to describe patient pathways, modeling approach, and analytic methods. We followed international ISPOR-SMDM and recent causal modeling guidelines.

Results: In the framework, 3 interventions for patients diagnosed with MSA were defined based on the MeDeMSA Care study design (NCT06072105): i) PBMC, ii) PBMC with TM, and iii) usual care (standard of care in the prospective European natural history MSA cohort). Health states were defined using the Unified Multiple System Atrophy Rating Scale Part IV (UMSARS_IV), ranging from 1 (completely independent) to 5 (bedridden) and incorporating both background and MSA-specific mortality. As agreed by clinical experts, the disease is well-described by a manageable number of disease states, therefore, a Markov state-transition cohort model was chosen. The simulated cohort starts at age 61, reflec-

ting early diagnosis, with a simulation cycle length of 6 months and a lifelong time horizon. Long-term outcomes include life expectancy, quality-adjusted life years (QALYs), harms, costs, and incremental cost-effectiveness ratios, and the model is constructed for cost-effectiveness and cost-utility analyses from societal and healthcare perspectives. Data from the European MSA Study Group (EMSA-SG) cohort were identified to inform the natural history and transition probabilities among the health states. Disease severity is represented through utility scores derived from the EQ-5D instrument. These utility scores are used to calculate QALYs accounting for the time in the respective health states for the 3 compared interventions. Resource use data are drawn from the clinical trial, with additional costs and harms informed by literature and public datasets.

Summary: This study presents an evidence-based decision-analytic framework for evaluating the long-term outcomes of best medical care for MSA. Future analyses, incorporating results from the MeDeMSA RCT, will inform clinical and healthcare system decisions to improve care for MSA patients.

P74: Suizidprävalenz und geschlechtsspezifische Unterschiede bei österreichischen Huntington-Patient*innen: Eine retrospektive Analyse

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Hintergrund: Patient*innen, die an der Huntington-Krankheit (HD) leiden, weisen im Vergleich zur Allgemeinbevölkerung eine deutlich höhere Prävalenz suizidalen Verhaltens auf. Die geschätzte Lebenszeitprävalenz von Suizidgedanken bei Huntington-Patient*innen liegt zwischen 20 % und 30,9 %. Suizid ist für 4,6–6,6 % der Todesfälle bei der Huntington-Krankheit verantwortlich und stellt damit die dritthäufigste Todesursache bei dieser Erkrankung dar.

Fragestellung: In dieser Studie wurde die Prävalenz von Suizid und suizidalem Verhalten zwischen der österreichischen Allgemeinbevölkerung und Huntington-Patient*innen, die von der Ambulanz der Universitätsklinik Innsbruck betreut wurden, verglichen.

Methodik: 91 zufällig ausgewählte Huntington-Patient*innen wurden retrospektiv ausgewertet, die in unserer Chorea-Spezial-Ambulanz behandelt werden. Alle neurologischen medizinischen Berichte dieser Patient*innen wurden analysiert. Todesfälle von an der Huntington-Krankheit leidenden Verwandten, die in der Familienanamnese dokumentiert und medizinisch bestätigt waren, wurden in die Analyse einbezogen. Nichtbestätigte Todesfälle wurden ausgeschlossen. Insgesamt wurden 127 Todesfälle registriert, davon 119 Fälle bestätigt und analysiert. Für den Vergleich mit der Allgemeinbevölkerung wurden die Daten zu Bevölkerung und Todesursachen für das Zensusjahr 2021 in Österreich herangezogen (91.962 Todesfälle). Es wurden der Chi-Quadrat-Test, der exakte Test nach Fisher und der Test für 2 unabhängige Proportionen verwendet.

Ergebnisse: Insgesamt wurden 119 Todesfälle bei Huntington-Patient*innen analysiert. Davon wurden 9 als Suizid oder Tod infolge eines Suizidversuchs klassifiziert. Die geschlechtsspezifische Auswertung ergab bei 5 von 62 (8 %) männlichen Verstorbenen und bei 4 von 57 (7 %) weiblichen Verstorbenen Suizid oder Tod infolge eines Suizidversuchs als Todesursache. Im Vergleich dazu gab es im Jahr 2021 in Österreich 91.962 Todesfälle, wovon 1.099 (1,2 %) Suizid oder Tod infolge eines Suizidversuchs als Todesursache hatten. Die geschlechtsspezifische Auswertung der österreichischen Gesamtbevölkerung ergab Suizid oder Tod infolge eines Suizidversuchs als Todesursache bei 879 von 46.010 (1,9 %) männlichen Verstorbenen und 220 von 45.952 (0,5 %) weiblichen Verstorbenen. Die Suizid-Prävalenz bei Huntington-Patient*innen zeigt sich im Vergleich zur Allgemeinbevölkerung signifikant höher, sowohl insgesamt ($p < 0,001$) als auch geschlechtsspezifisch (männliche Suizidfälle: Kontinuitätskorrektur: $p = 0,004$, Fisher: $p = 0,009$; weibliche Suizidfälle: $p < 0,001$). Diese Ergebnisse stimmen mit den Beobachtungen aus früheren internationalen Studien überein. Allerdings zeigte sich in der Huntington-Kohorte ein signifikant abweichendes Geschlechterverhältnis: Während in der Allgemeinbevölkerung etwa 4 männliche Todesopfer aufgrund von Suizid auf 1 weibliches Todesopfer entfallen, lag dieses Verhältnis in unserer Huntington-Kohorte bei 1,25:1 ($p < 0,001$).

Zusammenfassung: Unsere Ergebnisse zeigen eine signifikant höhere Suizidprävalenz bei Huntington-Patient*innen im Vergleich zur österreichischen Allgemeinbevölkerung, sowohl insgesamt als auch geschlechtsspezifisch. Auffällig ist das zur Gesamtbevölkerung abweichende Geschlechterverhältnis in der Huntington-Kohorte (1,25:1 vs. 4:1 in der Allgemeinbevölkerung), das auf krankheitsspezifische und psychosoziale Faktoren hinweist. Diese Befunde unterstreichen die Bedeutung gezielter Präventionsmaßnahmen und geschlechtsspezifischer Ansätze für diese Patient*innen.

P75: Sex differences in risk disclosure preferences for Parkinson's disease: An update of the Healthy Brain Aging (HeBA) Tirol study's risk disclosure analysis

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Background: Parkinson's disease (PD) is slightly more common in males than in females. Whether males and females differ in their views regarding risk disclosure for PD has not yet been investigated.

Objective: To analyse sex differences concerning risk disclosure preferences in an Austrian subcohort of the population-based, multicenter Healthy Brain Aging (HeBA) study.

Objective: To analyse sex differences concerning risk disclosure preferences in an Austrian subcohort of the population-based, multicenter Healthy Brain Aging (HeBA) study.

Method: After completion of an online PD risk assessment and at-home smell testing, a subset of participants underwent in-person visits with in-depth risk profiling as well as a structured risk disclosure questionnaire. Based on our pilot-project,[1] the initial questionnaire on risk disclosure preferences was adapted and 2 questions were added (5 multiple choice and 2 single choice questions). To examine sex differences, questions with scalable answer options were first analyzed by applying the Mann-Whitney-U test. Subsequently, differences in agreement with every individual answer option were analyzed utilizing chi-square/Fisher's exact tests. The significance level was set to $p < 0.05$ for the explorative non-parametric testing and adjusted according to Bonferroni for multiple testing in the second step of the analysis.

Results: This analysis includes the results of 153 participants (72 % female, median age 58 years) who completed the risk disclosure questionnaire between November 17 2023, and November 15 2024. Besides the high level of unconditional approval regarding disclosure of one's personal PD risk of 82 % (already shown in our pilot-study), a newly added question demonstrated that 72 % of the participants would like to be informed about their risk by a PD specialist. In the hypothetical scenario of a highly accurate positive PD test, 86 % of the participants stated to definitely or probably take part in clinical trials testing non-pharmacological treatments compared to 71 % in trials testing possible disease-modifying drugs.No statistically significant sex differences were observed regarding desire to be informed about one's personal risk, opinion on risk disclosure in general, and attitude towards participation in pharmacological clinical trials. However, females showed a higher willingness to participate in trials testing non-pharmacological interventions on a 4-point Likert scale ($p = 0.011$) and opted for more of the given choices that would be helpful for them in case of a positive PD test (median 4 for females vs. 3 for males, $p = 0.007$). In addition, women were more likely to choose to have follow-ups by a PD specialist (86 % female vs. 67 % male, $p = 0.012$) and to get in contact with support groups (42 % female vs. 19 % male, $p = 0.007$), but neither of these differences were statistically significant after Bonferroni correction.

Conclusion: Results of this study confirmed an open-minded and proactive attitude concerning PD risk disclosure in this Austrian HeBA study sample. Moreover, we could show sex differences in particular regarding dealing with one's risk. To draw practical conclusions, these results need to be replicated in larger and more diverse cohorts, for example in the entire European HeBA study cohort.

References:
[1] Mahlknecht P, Leiter S, Horlings C, et al. Preferences regarding Disclosure of Risk for Parkinson's Disease in a Population-based Study. Mov Disord Clin Pract. 2024 Nov 18. doi: 10.1002/mdc3.14264 [Online ahead of print]..

P76: Muscle mass, body fat mass, and nutrition in patients with Huntington's Disease

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Background: Huntington´s disease (HD) is an autosomal dominant, neurodegenerative disorder with characteristic motor, behavioral, and cognitive impairment. Mutant Huntingtin and its aggregates may also affect peripheral tissue causing muscle and adipose tissue wasting.
Objective: We aimed to assess skeletal muscle and fat mass, sarcopenia (according to the European Working Group on Sarcopenia in Older People 2 criteria), and nutrition in HD patients in different disease stages. We also aimed to compare body composition of HD patients with unrelated healthy controls.
Method: Bioelectrical impedance analysis was exploited to evaluate body composition in patients with symptomatic HD and age- and sex-matched healthy subjects. The Unified HD Rating Scale and cognitive assessments were used to characterize the patients clinically.
Results: Twenty HD patients (45 % female) with a median age of 57 years, body mass index of 22kg/m2, Total Motor Score of 17 points, and Total Functional Capacity of 10 points were included consecutively and prospectively. Confirmed sarcopenia (15 %, n = 3) was uncommon. Appendicular skeletal muscle mass index was reduced in 60 % (n = 12) and body fat mass in 35 % (n = 7). Muscle mass reduction was significantly associated with low weight (p = 0.049) and body fat (p = 0.048). Patients in disease stage 2 (n = 11) had a lower weight (p = 0.009) and body fat mass (p = 0.008) compared to patients in disease stage 1 (n = 9, i.e., patients without functional decline). Weight was also lower (p = 0.011) when compared to 20 healthy controls (45 % females) with a median age of 56 years. 55 % of HD patients were at risk for malnutrition or malnourished (Mini Nutritional Assessment). The latter correlated with weight (rs = 0.724, p < 0.001), skeletal muscle (rs = 0.473, p = 0.035), body fat mass (rs = 0.611, p = 0.004), motor symptoms (rs = -0.519, p = 0.019), independency (rs = 0.450, p = 0.046), and executive function (rs = 0.526, p = 0.017).
Conclusion: Reduction of muscle and fat mass seem to be common in HD patients, which may impact functional capacity and well-being.

P77: Verschreibung und Absetzen von Medikamenten bei der Huntington- Kohorte am Huntington-Zentrum der Universitätsklinik für Neurologie Innsbruck: Eine retrospektive Studie

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Hintergrund: Die Huntington-Krankheit (HK) ist eine autosomal-dominante neurodegenerative Erkrankung, die durch motorische, kognitive und psychiatrische Symptome gekennzeichnet ist. Eine kurative Therapie steht derzeit nicht zur Verfügung, und Empfehlungen zur symptomatischen Behandlung basieren häufig auf offenen Studien, Fallberichten und Expertenmeinungen. Unterschiede in Behandlungsstrategien je nach Krankheitsstadium, Geschlecht und Region sind entscheidend für die Entwicklung personalisierter Therapien. Für die Behandlung der Chorea sind in Österreich Tiaprid und Tetrabenazin zugelassen, wobei Antipsychotika bei psychiatrischer Komorbidität eine Alternative darstellen. Weitere Optionen mit eingeschränkter Evidenz sind Amantadin, Benzodiazepine oder Cannabis-Präparate. Die psychiatrischen Symptome werden symptomorientiert behandelt (z. B. Antidepressiva, Antipsychotika).
Fragestellung: Ziel der Studie war es, die im Huntington-Zentrum der Universitätsklinik für Neurologie Innsbruck verschriebenen Medikamente für neurologische und neuropsychiatrische Symptome der HK zu erfassen und die Gründe für das Absetzen oder Pausieren der Therapien zu analysieren. Ein besonderer Fokus lag auf den Abbruchraten der beiden zur Behandlung der Chorea in Österreich zugelassenen Medikamente, Tetrabenazin und Tiaprid.
Methodik: In einer retrospektiven Analyse wurden die Daten von 90 manifesten Huntington-Patient*innen (50 ▶

Frauen, 40 Männer) ausgewertet, die an der Chorea-Ambulanz der Universitätsklinik Innsbruck behandelt wurden. Medikationsdaten aus ambulanten und stationären Aufenthalten wurden nach Indikation und Abbruchgründen kategorisiert und deskriptiv analysiert. Der Vergleich der Abbruchraten von Tetrabenazin und Tiaprid aufgrund von Nebenwirkungen erfolgte mittels statistischer Tests für unabhängige Variablen.

Ergebnisse: Die am häufigsten verschriebenen Medikamentenkategorien bei Huntington-Patient*innen waren Antipsychotika (einschließlich Tiaprid) (n = 58), gefolgt von Antidepressiva (n = 56), Amantadin (n = 25), Tetrabenazin (n = 20) und Cannabinoiden (n = 12). Geschlechtsspezifische Unterschiede zeigten sich in der Verschreibung: Während Frauen am häufigsten Antidepressiva (n = 35 [70 %]) erhielten, wurden bei Männern vorrangig Antipsychotika (n = 24 [60 %]) verordnet. Antipsychotika (einschließlich Tiaprid) wurden zudem am häufigsten aufgrund von Nebenwirkungen (n = 39) abgesetzt. Ein Vergleich zwischen Tetrabenazin und Tiaprid ergab, dass Tetrabenazin signifikant häufiger als Tiaprid aufgrund unerwünschter Wirkungen abgesetzt wurde (Tiaprid n[abgesetzt] = 27[6], Tetrabenazin n = 20[12], p = 0,041).

Zusammenfassung: Die Ergebnisse unserer Auswertung zeigen, dass geschlechtsspezifische Unterschiede und Nebenwirkungen einen wesentlichen Einfluss auf die medikamentöse Behandlung der Huntington-Krankheit haben. Die höhere Abbruchrate von Tetrabenazin im Vergleich zu Tiaprid unterstreicht die Bedeutung einer individuellen Therapieanpassung, um Nebenwirkungen zu minimieren und die Lebensqualität zu verbessern.

P78: Age and sex differences in smell performance in a population-based study: Findings from the HeBA Tirol Cohort

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Background: Olfactory testing has proven to be one of the most important tools for the identification of subjects at risk for Parkinson's disease (PD), as olfactory dysfunction (OD) affects approximately 80 % of PD patients even years before motor symptom onset. These tests are quick to perform, cost-effective, and some—like the University of Pennsylvania Smell Identification Test (UPSIT)—can be performed remotely, making them suitable for large-scale population-based screenings. Previous studies have shown that the prevalence of OD in the overall population increases with age and is higher in males. Therefore, age- and sex-adjusted cut-off values are commonly applied. For the UPSIT, most of the data were gathered in North American cohorts[1] and population-based data for their validation in European cohorts are needed.

Objective: The objective of this study was to assess sex and age differences in the olfactory performance in the Healthy Brain Aging (HeBA) study that administered the UPSIT to a large Tyrolean cohort.

Method: In the present study, participants over the age of 50 with no prior PD diagnosis were invited to complete an online questionnaire including information on sex and age as well as simple PD risk factors. Subsequently, all online participants were invited to perform remote UPSIT testing. All participants with complete smell testing were included in the analysis and grouped according to sex and age categories. Group comparisons were performed for the raw UPSIT scores and the age- and sex-adjusted percentiles according to Brumm et al. Furthermore, analyses were conducted with binary data utilizing 2 cut-offs for hyposmia, according to the raw scores (< 25 out of 40) and the percentiles (≤ 9th).

Results: A total of 1514 participants with complete UPSIT testing were included in the analysis (60.4 % female, age 62.0 ± 7.7 years [mean ± SD]). Overall, mean UPSIT scores were 29.9 ± 4.5 and mean age- and sex-adjusted percentiles were 26.9 ± 21.1. Women had higher UPSIT scores than men (30.6 ± 4.2 vs. 28.8 ± 4.8; $p < 0.001$), while percentiles were lower in women (24.4 ± 21.0 vs. 30.7 ± 20.8; $p < 0.001$). Moreover, smell performance, according to the score, showed an age-dependent decline (age category 50–54 years: 31.2 ± 3.9; ≥ 80 years: 27.4 ± 4.5; $p < 0.001$), but percentiles showed an increase with age (age-category 50–54 years: 22.5 ± 21.9; ≥ 80 years: 44.9 ± 21.1; $p < 0.001$). Similar findings were obtained when binary data were analyzed. Participants who were female and younger were less likely to fall under the raw score cut-off of 25/40, but were more frequently labeled as hyposmic according to the percentiles.

Conclusion: Consistent with the literature, we found that UPSIT raw scores were higher in female participants and

decreased with age. However, when applying the age- and sex-adjusted cut-off values derived from North American cohorts, we observed an 'overadjustment' for age and sex, resulting in contrary outcomes. Therefore, these cut-offs may not be optimally suited for European cohorts and culturally adapted European age- and sex-adjusted percentiles are needed.

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P79: Sudden onset chorea and cognitive decline

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Background: Contactin-associated protein-like 2 (CASRP2) is a membrane protein found in the nervous system. It is part of the voltage-gated potassium channel complex (VGKCs) that modulates neuronal excitability and is therefore essential to prevent repetitive firing of nerves. Various autoimmune neurological disorders are associated with antibodies targeting proteins that form this channel. LGI1 is also a part of the VGKC, and as there is an overlap between these 2 proteins, antibodies against both might be observed. The clinical spectrum includes limbic encephalitis, epilepsy, Morvan syndrome, neuropathic pain, sleep disturbance, cognitive impairment, peripheral nerve hyperexcitability, as well as movement disorders.

Method: We report the case of a 78-year-old woman who presented to the emergency room with chorea that had been present for 1 month. The onset was described as sudden. Moreover, she described difficulties sleeping and showed some cognitive impairment on examination.

Results: Medication and family history were unremarkable. There was no indication of recent infectious disease. Subsequent examinations including cMRI and EEG showed no abnormal findings. A spinal tap showed a slightly elevated cell count, CXCL13 was 378pg/mL. No pathogenic agent was found. Chorea minor and neuroacanthocytosis were ruled out by blood sampling. Testing for paraneoplastic antibodies showed CASPR2 + antibodies. Tumour screening included gynaecological and dermatological examinations, CT thorax/abdomen, and a PET scan, all of which showed no evidence of a malignancy. A therapy with intravenous immunoglobulins (IVIG) was started immediately. After an initial clinical improvement, interval clinical evaluation again showed worsening of the symptoms, with increased chorea, falls, and further cognitive decline. Therefore, high dose short-term intravenous methylprednisolone was started and a plasmapheresis was performed. Unfortunately, the latter had to be stopped after 3 cycles due to urosepsis. IVIG was again given, this time with little clinical success, therefore rituximab (RTX) was started. Under RTX, the patient initially showed clinical improvement with resolution of chorea and mild cognitive improvement. At 2-month follow-up, the patient's condition had again deteriorated. Additionally, family members reported neuropsychiatric symptoms including recurrent binge eating. Flow cytometry revealed nearly complete depletion of CD19+ B-cells. Thus a 5-day course of IVIG was initiated, which resulted in mild clinical improvement.

Discussion: CASPR2 antibody-mediated autoimmune encephalitis can have a wide clinical presentation which also includes movement disorders. It is therefore an important differential diagnosis for new-onset movement disorders including chorea. It can show a good clinical response to immunosuppression, especially if treated early in the disease course. If the initial therapeutic response is insufficient, patients will benefit from fast escalation of therapy. Repeated screening for malignancies (i.e., every 6 months) will be required, although an association with an underlying tumour is more frequently occurring in autoimmune encephalitis linked to antibodies against intracellular antigens (e.g., Hu, Ri, Yo).

P80: Blood laboratory abnormalities in patients presenting at a memory clinic

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Background: Clinical dementia guidelines recommend blood laboratory tests in the diagnostic work-up for patients presenting with progressive cognitive symptoms. These tests are primarily advised to identify metabolic or hormonal blood abnormalities that may be (potentially reversible) causes of cognitive decline. However, the frequencies of blood abnormalities observed at the initial visit to a tertiary care memory clinic are poorly characterized.

AIM: To evaluate the prevalence of laboratory abnormalities in patients presenting at the memory clinic of the Department of Neurology of the Medical University of Vienna.

Method: We conducted a retrospective analysis of blood laboratory results (complete cell count, clinical chemistry, coagulation testing, folic acid, vitamin B12, cortisol, ceruloplasmin, TSH, anti-thyroid antibodies, thyreoglobulin, calcitonin, parathyroid hormone, homocysteine). The dataset comprised 1161 patients who presented at the memory clinic of the Department of Neurology (Medical University of Vienna) between September 2016 and June 2024, irrespective of their final diagnosis.

Results: XThe median age of the cohort was 72 ± 16 (IQR) years with no significant difference between females (73 ± 17) and males (71 ± 15). The most frequent abnormalities were a low relative lymphocyte count (41.76 % of all patients), followed by elevated total cholesterol (39.19 %) and LDL-cholesterol (30.75 %). Among changes associated with potentially reversible dementias, folic acid deficiency was most common (20.2 %), followed by hyperparathyroidism (9.26 %), hyponatremia (3.8 %), vitamin B12 deficiency (3.69 %), hypothyroidism (3.62 %), hypercalcemia (3.25 %), hyperthyroidism (2.6 %), iron deficiency (2.28 %), and hypoglycemia (0.41 %). In most cases, the abnormality was either not severe enough to be considered a plausible explanation for the cognitive deficit or the deficit was better accounted for by an alternative diagnosis.

Conclusion: Although blood tests are routinely recommended to rule out potentially reversible causes of dementia, only a small proportion of blood abnormalities qualified as such in our study. However, the routine use of blood laboratory testing can contribute to optimization of brain health. This is illustrated by our finding that one-third of our patients had insufficiently controlled levels of LDL-cholesterol, a factor which was recently recognized as a modifiable risk factor of dementia.

P81: Is transient global amnesia (TGA) really benign? A biomarker study

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Aim: To determine whether transient global amnesia (TGA) is associated with central nervous system (CNS) damage, as assessed by serum biomarkers neurofilament light chain (sNfL) and serum glial fibrillary acidic protein (sGFAP).

Method: In a prospective cohort of TGA patients, blood samples were obtained within 24–48 hours after TGA onset (t0) and 6 weeks thereafter (t1). We assessed sNfL and sGFAP levels using the highly sensitive single molecule array (Simoa) assay, and calculated z-scores adjusted for age, gender, and body mass index (BMI). Demographics, electroencephalography (EEG), and cerebral magnetic resonance imaging (cMRI) findings were also collected.

Results: A total of 20 patients were included (median age 66 years, 70 % female). No significant changes in sNfL or sGFAP levels associated with TGA at t0 and t1 were observed. Median sNfL z-scores were 0.45 (interquartile range [IQR] -0.09, 1.19) at t0 and 0.60 (IQR -0.61, 1.19) at t1. Median sGFAP z-scores were 0.27 (IQR -0.45, 0.76) at t0 and 0.44 (IQR -0.27, 0.75) at t1. Similarly, in the subgroup of patients with diffusion-weighted imaging (DWI)-positive hippocampal lesions (n = 5/20 [25 %]), no elevations in blood biomarkers were detected.

Conclusion: Our pilot study on neurological blood biomarkers supports the benign nature of TGA, indicating that no CNS tissue damage occurs.

P82: Identifying Alzheimer's dementia patients with new screening methods: Study design and first results of the ongoing Alzheimer-Check study

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Introduction: The diagnosis of Alzheimer's disease requires a time-consuming, complex, and resource-intensive process involving clinical assessment, MR-imaging, neuropsychological testing, PET-imaging, and/or lumbar puncture. In Austria, there is currently no standardized patient pathway and no systematic way of filtering patients, leading to an overburdening of specific healthcare sectors, long waiting times for patients, and a significant number of undiagnosed cases. With the arrival of new disease-modifying therapies, this issue will become even more critical, as timely diagnosis and treatment are essential for their effectiveness. A time- and resource-efficient screening method could be a practical way to identify individuals at high risk of Alzheimer's disease who require further diagnostic examinations.

Objective: The Alzheimer-Check study is a prospective, observational study that aims to evaluate a screening method using questionnaires and blood biomarkers to identify cognitively impaired individuals with intracerebral amyloid deposition.

Method: Participants are consecutively recruited through the memory clinic of the General Hospital of Vienna. Before their first visit to the memory clinic, they—and, if available, their caregivers—are asked to complete digital questionnaires on cognitive symptoms (Cognitive Change Index, CCI), functional performance (Functional Activity Questionnaire, FAQ), and depressive symptoms (Geriatric Depression Scale, GDS). After their visit, an additional blood sample is collected during the routine blood draw to analyze pTau217 levels. Other data points that will be considered for this study and used to assess the accuracy of the screening include a detailed neuropsychological examination, ApoE genotyping, amyloid PET, and/or lumbar puncture. These assessments will be conducted independently of this study. The recruitment goal for study participation is set at 250 participants.

Results: The study started in September 2024. Since then, 48 patients (mean age: 66.9 years, SD: 11.0; female: 25/48, 52.1 %) have been included. Of these, 11/48 (22.9 %) suffered from subjective cognitive decline, 23/48 (47.9 %) from mild cognitive impairment, and 13/48 (27.1 %) from dementia. One participant (2.1 %) had no cognitive symptoms. All patients included completed the digital questionnaires, and for 28/48 (58.3 %) participants, the caregiver also provided their responses. Plasma pTau217 values were analyzed for 34/48 (70.8 %) participants.

Conclusion: The combination of digital questionnaires and blood biomarkers could offer a resource-efficient screening method, helping to identify patients who need additional diagnostic assessment for Alzheimer's disease. In the future, such a screening method could be implemented in primary care to filter large numbers of patients and help to manage patient flow more effectively.

P83: Reduced cancer risk in patients with dementia even after accounting for surveillance bias: Results of a population-based cohort study

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Background: Several epidemiological studies have reported an inverse association between dementia and cancer risk. Whether this is due to an underlying biological mechanism or methodological bias is unclear. Surveillance bias is an example of the latter, describing reduced cancer screening in dementia patients as a cause of the lower cancer incidence.

Objective: This study aims to assess cancer incidence in a dementia cohort compared to a non-dementia cohort while accounting for surveillance bias.

Method: For this nationwide population-based cohort study, the dataset of the Austrian National Health Insurance Association was used, including dementia patients and age- and sex-matched non-demented individuals. For the control cohort, only data collected within the follow-up period of the matched subject in the dementia cohort were analyzed. Hazard ratios (HR) for cancer diagnosis were calculated using uni- ▶

variate and multivariate Cox regression models. To mitigate surveillance bias, a sub-analysis was performed, including only individuals who underwent diagnostic/imaging tests used for cancer screening (colonoscopy, gastroscopy, imaging of head, chest, and abdomen). **Results:** Overall, 340466 individuals were included in the study (170233 per study cohort). The mean age was 82.6 years (68.9–91.6), and the majority of the included subjects were female (225180/340466 (66.1 %). A total of 33513/340466 (9.8 %) individuals developed cancer during follow-up. Cancer risk in the dementia cohort was reduced compared to the control cohort, HR: 0.57 (95% CI: 0.55–0.58, $p < 0.001$). When including only individuals with diagnostic/imaging tests, this result remained significant for all analyzed cancer types, with the HR ranging from 0.29 (95% CI: 0.23–0.36, $p < 0.001$) for brain and meningeal malignancies to 0.71 (95% CI: 0.62–0.81, $p < 0.001$) for colon, rectal, and anal carcinomas. **Conclusion:** In our study, population dementia patients had a lower cancer risk than non-demented individuals. Consistent data from other studies support this finding. The inverse association is not attributable to surveillance bias, as it persists even when only patients with sufficient cancer screening are analyzed. To our knowledge, this is the first study that accounts for this bias. Limitations of this study include the lack of clinical data to validate diagnoses.

P84: Multiparametric analysis combining DSC-MR perfusion and [18F] FET-PET is superior to a single-parameter approach for differentiation of progressive glioma from radiation necrosis

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Background: Perfusion-weighted (PWI) magnetic resonance imaging (MRI) and O-(2-[18F]fluoroethyl-)-l-tyrosine ([18F]FET)-positron emission tomography (PET) are both useful for discrimination of progressive disease (PD) from radiation necrosis (RN) in patients with gliomas. Previous literature showed that the combined use of FET-PET and MRI-PWI is advantageous, however the increased diagnostic performances were only modest compared to the use of a single modality. Hence, the goal of this study was to further explore the benefit of combining MRI-PWI and [18F]FET-PET for differentiation of PD from RN. Secondarily, we evaluated the usefulness of cerebral blood flow (CBF), mean transit time (MTT) and time to peak (TTP) as previous studies mainly examined cerebral blood volume (CBV). **Method:** In this single center study, we retrospectively identified patients with WHO grade II–IV gliomas with suspected tumor recurrence, presenting with ambiguous findings on structural MRI. For differentiation of PD from RN we used both MRI-PWI and [18F]FET-PET. Dynamic susceptibility contrast MRI-PWI provided normalized parameters derived from perfusion maps (rCBV, rCBF, rMTT, rTTP). Static [18F]FET-PET parameters including mean and maximal tumor-to-brain ratios (TBRmean, TBRmax) were calculated. Based on histopathology and radio-clinical follow-up, we diagnosed PD in 27 and RN in 10 cases. Using the receiver operating characteristic analysis, area under the curve values were calculated for single- and multiparametric models. The performances of single and multiparametric approaches were assessed with analysis of variance and cross-validation. **Results:** After application of inclusion and exclusion criteria, we included 37 patients in this study. Regarding the in-sample based approach, in single parameter analysis, rTBRmean (AUC = 0.91, $p < 0.001$), rTBRmax (AUC = 0.89, $p < 0.001$), rTTP (AUC = 0.87, $p < 0.001$), and rCBVmean (AUC = 0.84, $p < 0.001$) were efficacious for discrimination of PD from RN. rCBFmean and rMTT did not reach statistical significance. A classification model consisting of TBRmean, rCBVmean, and rTTP achieved an AUC of 0.98 ($p < 0.001$), outperforming the use of rTBRmean alone, which was the single parametric approach with the highest AUC. Analysis of variance confirmed the superiority of the multiparametric approach over a single

parameter one (p = 0.002). While cross-validation attributed the highest AUC to the model consisting of TBRmean and rCBVmean, it also suggested that the addition of rTTP resulted in the highest accuracy. Overall, multiparametric models performed better than single parameter ones.

Summary: A multiparametric MRI-PWI and [18F]FET-PET model consisting of TBRmean, rCBVmean, and PWI rTTP significantly outperformed the use of rTBRmean alone, which was the best single parameter approach. Secondarily, we firstly report the potential usefulness of PWI rTTP for discrimination of PD from RN in patients with glioma. However, for validation of our findings, prospective studies with larger patient samples are necessary.

P85: Efficacy and safety of daridorexant in patients with chronic insomnia disorder and comorbid nocturia

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Background: Chronic insomnia and nocturia are frequently associated, particularly in older adults, and both conditions significantly impair sleep quality, daytime functioning, and quality of life. Despite the high prevalence of nocturia-related sleep disruptions, few studies have evaluated the efficacy of hypnotics in improving both sleep and nocturia symptoms. Daridorexant 50mg has demonstrated efficacy in improving sleep maintenance and daytime functioning without next-day residual effects in patients with chronic insomnia.

Objective: This study aimed to evaluate the efficacy and safety of daridorexant 50mg compared to placebo in patients with chronic insomnia and comorbid nocturia.

Method: This double-blind, placebo-controlled crossover study randomized patients aged 55 years or older, with an insomnia severity index score of ≥ 13 and a history of at least 3 nocturnal voids per night for over 1 month, to receive either daridorexant 50mg or placebo for 4 weeks. After a washout period of 14–21 days, patients switched to the alternate treatment for an additional 4 weeks. Efficacy endpoints included changes from baseline in self-reported total sleep time (sTST), insomnia severity index score, quality and depth of sleep assessed via visual analogue scale, number of nocturnal voids, and time to first nocturnal void. Daytime functioning was measured using the Insomnia Daytime Symptoms and Impacts Questionnaire. Safety endpoints included the incidence of adverse events

Results: Daridorexant significantly increased sTST compared to placebo at week 4, with a least-squares mean difference of 20.9 minutes (95% CI: 8.0, 33.7; p = 0.002). Improvements in sTST were observed from week 1 and sustained throughout treatment. Quality and depth of sleep improved significantly with daridorexant vs. placebo at all time points except for the week 4 visual analogue scale for sleep quality, where the difference was not statistically significant (p = 0.058). Insomnia severity scores decreased significantly with daridorexant at weeks 2 and 4. Daridorexant also reduced the number of nocturnal voids compared to placebo and increased the median time to first void, with significant differences observed at week 1 and sustained improvements at week 4. A greater proportion of patients treated with daridorexant reported fewer than 2 voids per night at weeks 1 and 4. Daytime functioning, assessed via Insomnia Daytime Symptoms and Impacts Questionnaire scores, improved over time with both treatments, with greater improvements observed for daridorexant. No serious adverse events or treatment discontinuations occurred, and no falls or urinary incontinence were reported with daridorexant.

Conclusion: Daridorexant 50mg significantly improves sleep duration, quality, and daytime functioning in patients with chronic insomnia and comorbid nocturia. Additionally, daridorexant reduces nocturia symptoms, including fewer nocturnal voids and a longer time to first void, without increasing the risk of falls or urinary incontinence. These findings highlight the potential of daridorexant as a clinically relevant treatment option for this challenging patient population.

P86: Effect of daridorexant on sleep architecture in adult patients with insomnia disorder: An analysis of two pooled phase 3 studies

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Introduction: Insomnia is a prevalent sleep disorder, with hyperarousal considered a central pathophysiological factor. Analyzing sleep microarchitecture, particularly spectral components and sleep spindles, along with sleep continuity assessed via polysomnography (PSG), provides insights into the hyperarousal observed in insomnia. Orexin-producing neurons—key to wakefulness, sleep/wake transitions, and stress—may be hyperactivated in insomnia. This analysis assesses whether daridorexant, a dual orexin receptor antagonist, modifies sleep architecture, with a focus on hyperarousal patterns such as elevated fast frequencies and disrupted sleep continuity, using pooled data from two phase 3 trials.

Method: Data from two phase 3 trials investigating daridorexant treatment (10mg, 25mg, and 50mg) in adults with insomnia disorder were analyzed. Participants met DSM-5 criteria for insomnia, experiencing sleep onset, maintenance issues, or early awakenings for at least 3 months. This analysis included participants with available PSG data: placebo (n = 586), daridorexant 25mg (n = 584), and daridorexant 50mg (n = 296). Electroencephalogram (EEG) microarchitecture was analyzed across spectral bands: delta (0.5–4Hz), theta (4–8Hz), alpha (8–12Hz), and beta (12–30Hz). Sleep-wake stages were determined, and sleep spindles (11–15Hz) in N2 were assessed. A linear mixed-effects regression model was used to analyze the effects of daridorexant on power spectral analysis and sleep stage transitions.

Results: Daridorexant 50mg significantly reduced the probability of transitioning from wakefulness to wakefulness at month 3 (-6.9 %) and increased transitions from wakefulness to N1, N2, and REM stages. Similar, though less pronounced, results were observed at month 1 with daridorexant 25mg. At month 3, daridorexant 50mg significantly reduced relative alpha power (-1.7 %) and increased relative delta power (+2.9 %) during wakefulness compared to placebo. Additionally, relative beta power was significantly reduced during wakefulness and N1 at month 3. No significant differences were seen in N2, N3, and REM stages between treatment groups. No effects on sleep spindles were observed.

Conclusion: Daridorexant 25mg and 50mg treatment for 3 months facilitated quicker transitions to sleep, as shown by a decreased probability of sustained wakefulness. Daridorexant 50mg significantly reduced relative alpha and beta spectral power, markers of wakefulness. No significant effects on sleep spindles were found. These findings suggest that daridorexant may reduce hyperarousal associated with insomnia.

P87: Effect of daridorexant on wakefulness throughout the night and morning sleepiness in patients with insomnia disorder

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Background: Reducing wakefulness throughout the entire night without residual effects the next morning is an essential characteristic of drugs for chronic insomnia. Daridorexant, a dual orexin receptor antagonist, is approved for the treatment of chronic insomnia. In phase 3 studies, daridorexant significantly reduced polysomnography (PSG)-determi-

ned wake after sleep onset (WASO). However, analyses of its effects by quarter of the night and correlations between nighttime and daytime endpoints are lacking.

Objective: To examine the effect of daridorexant on WASO in each quarter of the night, self-reported morning and daytime symptoms, and the correlation between nighttime and daytime measures in patients with chronic insomnia over a 3-month treatment period have been analyzed.

Method: This analysis used data from a phase 3, randomized, double-blind, placebo-controlled study with 930 patients (daridorexant 50mg: n = 310; 25mg: n = 310; placebo: n = 310). Eight-hour PSG recordings were conducted during the single-blind placebo run-in (baseline) and at months 1 and 3 of treatment. WASO, defined as the cumulative time (minutes) spent awake after sleep onset, was analyzed by each quarter of the night (Q1–Q4). Daily sleep diaries captured visual analogue scale (VAS) scores for morning sleepiness, daytime alertness, and ability to function. Changes from baseline were assessed using linear mixed-effects models. Correlations between WASO and VAS scores were calculated using random-effects models.

Results: Daridorexant significantly reduced WASO in Q2, Q3, and Q4 compared to placebo at months 1 and 3, with progressive reductions over the night, particularly with 50mg. Greater reductions in WASO were observed with 50mg compared to 25mg. Morning and daytime VAS scores for sleepiness, alertness, and functioning improved in a dose-dependent manner from baseline, with statistically significant effects of 50mg compared to placebo from week 2 onwards. Strong positive correlations were observed between self-reported VAS scores, but no clinically significant correlations were found between WASO by quarter of the night and VAS scores.

Conclusion: In patients with chronic insomnia, daridorexant significantly reduced WASO throughout the night, and independently improved morning and daytime functioning and alertness. Given the unmet need for sleep-promoting drugs that maintain efficacy throughout the night without next-day sedation, these findings are clinically relevant.

P88: Non-motor prodromal multiple system atrophy: A systematic review

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Background: The 2022 Movement Disorder Society (MDS) criteria for the diagnosis of multisystem atrophy (MSA) introduce the research category of possible prodromal MSA. This consists of the presence of isolated, otherwise unexplained, cardiovascular or urogenital autonomic failure (iAF), or isolated rapid-eye-movement sleep behavior disorder (iRBD), accompanied by subtle cerebellar and parkinsonian signs that may precede the onset of MSA motor symptoms by months or years.

Research question: Aiming to explore the prodromal phase of MSA by focusing on its non-motor presentations (iAF or iRBD), we compared the total and annual phenoconversion rate to neuronal (Parkinson's disease [PD] or dementia with Lewy bodies [DLB]) or glial alfa-synucleinopathies MSA in individuals with iAF or iRBD. We further assessed biomarkers able to discriminate individuals retaining a "pure" iAF or iRBD phenotype from phenoconverters and to predict the final phenotype.

Method: The literature search was performed in Pubmed database by using the following combination of keywords: "Parkinson's disease," "PD," "multiple system atrophy," "MSA," "dementia with Lewy-bodies," "DLB," "isolated autonomic failure," "iAF," "lower urinary tract symptoms," "LUTS," "erectile dysfunction," "ED," "isolated rapid eye movement sleep behavior disorder," "iRBD." Only original articles in English providing information on phenoconversion rates to glial or neuronal synucleinopathies were included. Data on phenoconversion rate to MSA and PD/DLB was extracted from the selected papers by using a structured spreadsheet. The rate of phenoconversion from iAF, LUTS, ED, or iRBD to either MSA, PD, or DLB was weighted against the sample size of each dataset and an annual conversion rate was calculated to account for the different follow-up periods.

Results: The search resulted in 3040 articles, of which 439 were removed because they were duplicates and 2601 articles were screened by title or abstract. 136 articles were selected for full-text reading, and 58 were chosen for the final analysis. We found that the average annual conversion rate from iAF to MSA was 2.72 % and 2.65 % to PD or DLB. In people with iRBD, the average yearly phenoconversion rate was 0.5 % to MSA and 7.55 % to PD or DLB. In retrospective MSA cohorts, the first MSA symptom was erectile dysfunction in 28 % of males and LUTS in 25 % of cases on average.

Conclusion: The findings highlight the importance of early recognition and characterization of prodromal MSA, ▶

as well as the challenges in distinguishing it from prodromal PD and DLB. It seems crucial to consider atypical parkinsonian syndromes, like MSA, especially in people with iRBD, while in people with iRBD the phenoconversion to PD or DLB seems to be more frequent. Further research incorporating larger prospective studies and standardized diagnostic criteria is warranted to improve the understanding and management of prodromal MSA.

P89: Telemedicine in multiple system atrophy: The MeDeMSA Care feasibility assessment

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Background: The telemedicine momentum following the COVID-19 pandemic promoted the delivery of healthcare services to people in their own homes. In multiple system atrophy (MSA), telemedicine may ensure continuity of care, particularly when the disease progresses and physical and geographical barriers arise.

Objective: to assess the feasibility of telemedicine-based procedures including neurological consultations, psychological counseling, physiotherapy, speech therapy and occupational therapy for individuals with MSA, and to identify barriers to such approaches.

Method: This feasibility assessment is embedded in the prospective MeDeMSA Care study. The enrolled subjects were randomly assigned 1:1 to 2 arms, 1 receiving standard medical care with in-person visits every 6 months, the other receiving standard medical care plus monthly remote consultations with different healthcare providers on the CHES (Computer-Based Health Evaluation System) online platform of the Innsbruck University Hospital. At the time of recruitment, healthcare providers assessed the feasibility of telemedicine visits using a standardized questionnaire with numeric rating scales (NRS) and a list of potential barriers. These evaluations were then repeated at the end of each telemedicine visit to report on the feasibility assessed and the barriers encountered. The individuals with MSA were asked, after each follow-up visit, to complete online satisfaction surveys entailing a NRS and 3 open questions to identify barriers and benefits of this novel treatment strategy.

Results: To date, 20 individuals with MSA (60 % female; 50 % MSA type-P) were enrolled, 10 of whom (4 female; 50 % MSA type-P) were randomized to the telemedicine arm. The MeDeMSA Care team performed 297 remote visits. The telemedicine feasibility was rated as 7.7/10 (SD: 2.2) on the NRS by the healthcare providers, who reported the closer monitoring (n = 50/297 visits) and the presence of the caregiver (n = 18/297 visits) as benefits of the telemedicine, and the presence of technological challenges (n = 68/297 visits) and disease progression (including worsening of motor skills and dysarthria; n = 47/297) as major barriers. Individuals with MSA filled out 249 satisfaction surveys with an NRS average satisfaction score of 8.4/10 (SD: 1.7). The closer monitoring (reported by 8/10 enrolled subjects) and the multidisciplinary care (n = 5/10) were described as telemedicine benefits. Concerning barriers, 4/10 individuals with MSA reported technology-related difficulties, but only 1 mentioned the presence of disease-related barriers.

Conclusion: Telemedicine has the potential to deliver neurological, psychological, and neuro-rehabilitation support to individuals with MSA throughout the disease course. However, telemedicine platforms and devices should be constantly upgraded to align to users' motor- and age-related technological abilities.

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P90: Physical counterpressure maneuvers biofeedback training in postural orthostatic tachycardia syndrome: A monocentric, randomized controlled trial (RCT)

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Background: Based on clinical experience, expert recommendations suggest the use of counterpressure maneuvers (CPM) in individuals with postural orthostatic tachycardia syndrome (POTS). However, evidence of the efficacy and safety of CPM to improve orthostatic tolerance or hemodynamic responses in POTS is missing at this time.

Research question: The present study evaluates whether performing 14-day CPM-biofeedback training improves the symptomatic burden (primary objective) and secondarily the interference of POTS symptoms with daily activities, fatigue, and health-related quality of life of individuals with POTS compared to best clinical practice non-pharmacological measures. Secondary in-laboratory objectives are to assess the influence of CPM on the supine-to-standing heart rate (HR) and blood pressure (BP) changes as well as on the severity of orthostatic intolerance after performing CPM for 2 minutes compared to a baseline (intervention-free) active standing test, and to assess the safety and tolerability of CPM-biofeedback training in individuals with POTS.

Method: This is a monocentric, proof-of-concept, 1:1 randomized controlled trial with rater-blinded evaluation of the hemodynamic effect of CPM in 40 individuals suffering from POTS. The trial foresees 3 study visits for both the interventional and the control arm (screening and baseline visit on-site, as well as a telephone visit 14 days later). All study participants will receive detailed counselling on CPM and other behavioral and non-pharmacological measures to combat POTS symptoms in daily life (best clinical practice). Participants randomized to the interventional arm will receive a CPM-biofeedback training session in the autonomic function laboratory at the Department of Neurology of the Innsbruck Medical University to learn 4 different CPM under continuous HR and BP monitoring, and an additional day-7 telephone visit. To evaluate the baseline to day 14 change in symptom severity, the Malmö POTS Score (MAPS) total score (primary endpoint) and—for secondary endpoints—the MAPS single items, Vanderbilt Orthostatic Symptom Score, Orthostatic Grading Scale, Fatigue Severity Scale, and Health-related Quality of Life Questionnaire will be administered. We further evaluate the following secondary in-laboratory endpoints in participants randomized to the interventional CPM-biofeedback arm: Severity of orthostatic intolerance (on a 0–10 points scale) at the end of the 4 different CPM practiced upon standing (CPM1–4) as well as hemodynamic changes (HR, systolic and diastolic BP) during the CPM practiced upon standing (CPM1-4) compared to the baseline active standing test.

Results: Recruitment starts in January 2025 and is expected to be completed within 12 months.

Conclusion: The study will provide evidence for the non-pharmacological treatment of POTS patients.

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P91: Cardiovascular autonomic disorders following COVID-19 infection or vaccination: The Innsbruck experience

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Background: Neurological manifestations, including symptoms reminiscent of autonomic dysfunction, may frequently occur in individuals with COVID-19. Cardiovascular autonomic disorders have been reported after both COVID-19 infection and vaccination, but previous reports were limited to single cases or small series with poor diagnostic accuracy or limited follow-up.

Aim: To investigate the type and frequency of new-onset cardiovascular autonomic disorders (CAD) following COVID-19 infection or vaccination in a retrospective case series from the Innsbruck Dysautonomia Center. In identified cases, we studied the frequency of associated autonomic and non-autonomic complaints, applied treatment, and clinical outcome at last available follow-up.

Method: Medical records of individuals referred to the Innsbruck Dysautonomia Center between March 2020 and March 2023 were reviewed for new-onset of orthostatic intolerance (OI) or recurrent syncope within 6 weeks of a passed COVID-19 infection or vaccination. Frequencies were analyzed with the Pearson's Chi-square, Fisher's exact, or Fisher-Freeman-Halton test where appropriate. Depending on the data distribution, differences in quantitative variables were assessed with the Student's T or Mann–Whitney U test. Statistical significance was set at a 2-tailed p-value of < 0.05, with a block-wise Bonferroni correction being applied for multiple testing.

Results: By screening 1081 individuals referred to our center between March 2020 and March 2023, we identified 101 cases with new-onset of OI or recurrent syncope within 6 weeks of a passed COVID-19 infection (n = 75) or vaccination (n = 26). Following COVID-19 infection, 21 (28 %) individuals were newly diagnosed with postural orthostatic tachycardia syndrome (POTS), 12 (16 %) with vasovagal syncope (VVS), 1 (1.5 %) with delayed and 1 (1.5 %) with transient orthostatic hypotension (OH), while in 40 (53 %) cases with OI, the diagnostic work-up excluded any CAD (OIw/oCAD). Post-COVID POTS cases were younger (33 ± 9 vs. 44 ± 14 years; p = 0.002), more frequently vaccinated against COVID-19 (79 % vs. 42 %; p = 0.011), and reported additional autonomic complaints more often (100 % vs. 67 %; p = 0.008) than post-COVID OIw/oCAD.

Following COVID-19 vaccination, 11 (42 %) POTS, 2 (8 %), VVS and 3 (12 %) transient OH cases were newly diagnosed, while 10 (38 %) cases were classified as OIw/oCAD. Post-vaccination POTS cases were younger (32 ± 10 vs. 47 ± 14 years; p = 0.009) and more frequently reported thermoregulatory complaints (100 % vs. 25 %; p = 0.002) compared to post-vaccination OIw/oCAD.

Additional non-autonomic complaints were common in both the post-COVID and post-vaccination cohort, led by fatigue, neurocognitive complaints, and headache (in descending order), without significant differences observed at group-wise comparison. Lifestyle modifications and non-pharmacological measures were recommended to all individuals diagnosed with CAD following COVID-19 infection or vaccination, with one third additionally receiving pharmacological treatment. Follow-up was available in 42 (82 %) individuals with post-COVID and post-vaccination CAD, with a symptomatic improvement observed in 26 (62 %) cases.

Summary: A specialized diagnostic work-up is pivotal to diagnose or exclude CAD in individuals with new-onset OI or recurrent syncope following COVID-19 infection or vaccination. POTS was the most common new diagnosis; however, in half of the referred cases, cardiovascular autonomic function testing excluded any CAD. In the experience of the Innsbruck Dysautonomia Center, a multimodal treatment approach resulted in symptomatic improvement for a substantial proportion of affected individuals.

P92: Autonomic history-taking and escalating patient care to a specialized ANS outpatient clinic

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Background: In 2021 Thijs and colleagues, on behalf of the European Federation of Autonomic Societies (EFAS), published recommendations regarding how to approach a patient with transient loss of consciousness (TLOC). In 2024, a 68-year-old female with parkinsonism was referred from an external Movement Disorder Outpatient Clinic to our specialized ANS outpatient clinic in University Hospital Tulln. The patient was strongly suspected to have multisystem atrophy parkinsonian type (MSA-P); however, the clinical presentation was atypical.
Aim: With the patient case presented, we expand on the recommendations of Thijs et al. to aid neurologists, neurological sub-specialists, practical physicians, and internal medicine specialists in their clinical decision making when deciding whether to refer their patients to a specialized ANS outpatient clinic.
Method: Following the EFAS recommendations, a complete neurological exam, detailed autonomic history, and complete tilt table test battery were conducted.
Results: Symptoms began in 2021 and slowly progressed, also involving urinary incontinence and vertigo. A (prolonged) trial of Levodopa was not tolerated by the patient and yielded no clinical benefit. In light of repetitive falls, a Schellong test was performed and was normal. She reported reduced sweating, dry mouth and eyes, and skin colour changes. The tilt table testing was conducted with all medications (including 40mg propranolol) taken. An expiratory-inspiratory ratio of 0.99 (normal value [NV] 1.10), blocked phase IIb, Valsalva ratio of 1.09 [NV 1.16], and 30:15-ratio of 1.05 [NV 1.06]) were found. A progressive fall in blood pressure (BP) of over 30mmHg systolic over 15 minutes after tilting and BP fall of over 30mmHg systolic after standing (recovery after 40 seconds, respective 30:15-ratio of 0.99) were recorded. Under pharmacological influence, the tilt table findings revealed evidence of abnormal parasympathetic and mildly abnormal sympathetic function, with additional evidence of asymptomatic delayed orthostatic hypotension. .
Conclusion: The case discussed exemplifies a point in patient care where a specialized ANS work-up becomes necessary. MSA-P patients may not present with predominant orthostatic autonomic symptoms initially. Careful examination and history may prompt expert autonomic testing, even if a standard Schellong or orthostasis test is normal. Hence, this case highlights the use of specialized ANS clinics, the importance of documenting all domains of autonomic function in extrapyramidal syndromes, and – when possible – support of the diagnosis with additional autonomic testing.

P93: The International Dysphagia Diet Standardization Initiative (IDDSI) – Hintergründe und Implementierung einer weltweiten Standardisierungsinitiative texturmodifizierter Kostformen bei (neurogenen) Dysphagien

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Hintergrund: Konsistenzveränderungen von Speisen und Getränken stellen eine weit verbreitete Strategie in der Versorgung von Patient*innen mit neurogenen Schluckstörungen (Dysphagien) dar. Bislang basieren die Grundlagen, Terminologie und Testbarkeit diätetischer Kostformen nicht auf einer interdisziplinär fundierten Evidenz, was sowohl die Sicherheit und Autonomie der Patient*innen gefährdet als auch zu Missverständnissen und unklaren Empfehlungen auf intra- und interinstitutioneller Ebene führt. Die Entwicklung der International Dysphagia Diet Standardization Initiative (IDDSI) ermöglicht eine standardisierte, auf schluckphysiologischen Aspekten ▶

basierende, 8-stufige Klassifikation und Testung von konsistenzmodifizierten Kostformen (Cichero et al., 2017). Weltweit ist der Implementierungsprozess in mehr als 50 Ländern im Gange, und internationale Fachgesellschaften empfehlen die Umsetzung. Die Implementierung der IDDSI in klinischen Einrichtungen der DACH-Region steht dabei vor verschiedenen Herausforderungen. Es existiert jedoch nur eine spärliche Datengrundlage, die den Implementierungsprozess auf (inter)nationaler Ebene überwacht und Variablen identifiziert, welche die Akzeptanz des Grundgerüsts im klinischen Kontext fördern.

Fragestellung: Eine zentrale Frage lautet daher, welche Faktoren die erfolgreiche Implementierung der IDDSI in klinischen Einrichtungen der DACH-Region beeinflussen und wie sich strukturelle Rahmenbedingungen sowie interdisziplinäre Aspekte auf die Umsetzung der Initiative auswirken.

Methode: Der Vortrag beleuchtet die schluckphysiologische Motivation texturmodifizierter Kostformen und zeigt die globalen Fortschritte der IDDSI auf. Auf Basis bestehender quantitativer (Fragebogenerhebungen) und qualitativer (interdisziplinäre Expert*-inneninterviews) Vorarbeiten sowie vorläufiger neuer Daten werden der aktuelle Wissens-, Planungs- und Implementierungsstand für Österreich, Deutschland und die Deutschschweiz zusammengefasst und förderliche sowie hinderliche Faktoren für eine erfolgreiche Implementierung identifiziert.

Ergebnisse: 2019 konnte für Österreich eine Datengrundlage für 75 neurologische Akut- und Langzeiteinrichtungen zum Implementierungsstand der IDDSI gesammelt werden. Dies wird durch aktuelle Erhebungen in Österreich (n = 84), Deutschland (n = 186) und der Deutschschweiz (n = 38) erweitert, die unterschiedliche Förder- und Hemmfaktoren aufgrund von Rechtsform, Versorgungsstufe und Verpflegungsform identifizieren. Qualitativ zeigen sich aus interdisziplinärer Sicht die Präsenz eines Projektteams, finanzielle Planungssicherheit und Schulungsaspekte als kritische Themen einer erfolgreichen Einführung.

Zusammenfassung: Die International Dysphagia Diet Standardization Initiative (IDDSI) bietet eine weltweit anerkannte, standardisierte Klassifikation für die Texturanpassung von Nahrungsmitteln, um die Sicherheit und Autonomie von Patient*innen mit Schluckstörungen zu verbessern. Vorläufige Ergebnisse zeigen, dass strukturelle Rahmenbedingungen und interdisziplinäre Themen maßgeblich den Erfolg der Implementierung beeinflussen.

Literatur:
- Cichero JAY et al. (2017). https://doi.org/10.1007/s00455-016-9758-y

P94: A mixed methods study on the emergence of nonverbal and heart-rate synchrony during single music therapy sessions in neurorehabilitation

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Background: Recent advancements in neuroimaging have increasingly linked neurology and music therapy. Music therapy has been shown to enhance functional abilities in neurorehabilitation, including gait, upper extremities, speech, and cognition. This study aimed to understand the mechanisms of music therapy by focusing on the behavioral and relational aspects, specifically dyadic nonverbal synchrony (NVS) and physiological heart-rate synchrony (HRS), between patients and therapists. NVS, linked to empathy, is quintessential for building a positive therapeutic relationship. Higher NVS correlates with improved relational quality, self-efficacy, and symptom reduction. HRS is associated with therapeutic progress, occurring at empathetic moments, suggesting a connection between heart-rate synchrony and shared emotional states. This study explored the emergence of NVS and HRS during music therapy in neurorehabilitation and identified key characteristics of dyadic interactions.

Method: In this mixed-methods study, 11 inpatients without verbal or physical limitations participated with a music therapist in single sessions at a neurorehabilitation facility in Lower Austria. As the choice of instruments would predetermine the movements, NVS was compared during the non-structured conversations 2 minutes before and after the music intervention. The data was quantified and determined (10 sec segments, ± 2 sec lag) with the applications Motion Energy Analysis (MEA) and rMEA. Heart rate was sampled with electrocardiogram sensors at 1000Hz, and HRS was quantified and determined (30 sec segments, ± 2 sec lag) using rMEA. Subsequently, qualitative content analysis was adopted to examine the therapeutic interaction during the 4 highest and lowest HRS segments.

Results: The analysis revealed that NVS post-intervention was significantly above chance, and a significant increase in both NVS and patient leading. Similarly, HRS during the music intervention exceeded chance and positively

correlated with its duration, indicating an optimal duration of 25 minutes. Differences in therapeutic interactions were observed between high and low HRS segments. In high HRS segments, patients showed greater empowerment and external awareness, dyads listened more to each other and demonstrated increased relaxation, connection, and physical attunement.

Conclusion: Music intervention seemed to have effectively increased NVS and promoted patient empowerment, as reflected by the increased patient leading, which is a crucial factor for reducing drop-out rates. The differences observed in HRS may be attributed to variations in the dyadic interactions. Given the importance of patient participation in neurorehabilitation, a high HRS is thus beneficial, hence the importance of the duration of music intervention. This study explored the innovative application of NVS and HRS analysis within a neurorehabilitation setting, which may prove useful in future research involving patients with verbal or physical limitations, such as those with disorders of consciousness. Moreover, this study underscored the significance of the music intervention duration, an important aspect of health policy that impacts both patients and clinicians.

P95: Neurofilament light chain predicts acute mountain sickness, erythropoietin predicts neuroaxonal damage in simulated high altitude

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Background: Neurological symptoms are common in acute mountain sickness (AMS). However, the extent of neuroaxonal damage remains unclear. Neurofilament light chain (NfL) is an established blood biomarker for neuroaxonal damage, and erythropoietin (EPO) plays a crucial role in the early adaption to high altitude and is possibly involved in neuroprotection.

Objective: To investigate whether i) plasma (p) NfL levels increase after simulated altitude exposure; ii) pNfL levels correlate with the occurrence of AMS; iii) pNfL increase might be mitigated by pre-acclimatization; and iv) EPO dynamics are linked to neuroaxonal damage as measured by NfL.

Method: Healthy subjects were exposed to simulated high altitude (4500m) by the use of a normobaric hypoxic chamber at the University of Innsbruck, 2 times, i.e., within cycle 1 (C1) over 12 hours, and within cycle 2 (C2) for another 12 hours. Prior to C2, subjects were randomly assigned to prior acclimatization or sham acclimatization for one week. Before each cycle (measurement [M] 1 and 3) and after each cycle (M2 and M4), clinical data (arterial oxygen saturation [SaO_2], heart rate, and Lake Louise AMS score [LLS]), and plasma samples were collected. pNfL was measured using the single molecule array (Simoa) technique, EPO was quantified using commercially available ELISA kits.

Results: pNfL levels did not significantly change within each study cycle, however they increased over the total study period (M1: 4.57 [3.34–6.39], M2: 4.58 [3.74–6.0], M3: 5.64, M4: 6.53 [4.65–7.92] pg/ml, $p < 0.001$). EPO levels already increased in C1 (M1: 4.17 [2.99–5.67] vs. M2: 10.12 [7.86–14.06] mU/ml, $p < 0.001$). Subjects suffering from AMS during the study procedures showed higher pNfL levels and NfL z-scores at M4 (pNfL: 6.80 [6.19–8.13] vs. 5.75 [4.17–7.35], $p = 0.048$; NfL z-scores: 1.02 [0.62–0.50] vs. 0.25 [-0.95–1.08], $p = 0.011$) and a higher total pNfL and NfL Z score increase (pNfL: 2.88 [1.21–3.48] vs. 0.91 [0.53–1.48], $p = 0.022$; 1.56 [0.53–2.45] vs. 0.78 [0.32–1.00], $p = 0.072$) compared to subjects without AMS, even if the NfL z-score increase did not reach statistical significance. An effect of pre-acclimatization on pNfL levels or NfL z-scores could not be observed. Subjects with high EPO levels showed significantly lower NfL concentrations (5.85 [4.15–6.85] pg/ml vs. 6.73 [4.70–8.64] pg/ml, $p = 0.030$) as well as lower NfL z-scores (0.64 [-0.88–1.17] vs. 0.95 [0.25–1.48], $p = 0.040$) than those with low EPO levels.

Conclusion: PNfL increases alongside exposure to simulated altitude and is associated with AMS. Higher EPO concentrations were associated with lower NfL levels. This might further advocate for the hypothesis of a neuroprotective role of EPO.

P96: Diagnostik, Therapie und Monitoring bei idiopathischer intrakranieller Hypertension – Konsensus-Empfehlungen des österreichischen IIH-Netzwerks (AN4IH)

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Hintergrund: Diagnostik, Therapie und Monitoring von Patient*innen mit idiopathischer intrakranieller Hypertension (IIH, früher auch bekannt als „Pseudotumor cerebri" oder „benigne intrakranielle Hypertension") zeichnen sich durch ein hohes Maß an Komplexität aus und erfordern die interdisziplinäre Zusammenarbeit zwischen Neurologie, Ophthalmologie, Neuroradiologie, Neurochirurgie und Endokrinologie. Daher empfehlen die maßgeblichen internationalen Leitlinien, dass Diagnostik, Therapie und Monitoring an spezialisierten Zentren erfolgen sollen, die eine entsprechende interdisziplinäre Struktur vorhalten können. In Österreich gibt es dafür bisher keine standardisierte Versorgungsstruktur, um diesen Anforderungen gerecht zu werden.
Ziel des Austrian Network for Idiopathic Intracranial Hypertension (AN4IH) ist es, ein entsprechendes Kompetenznetzwerk zu schaffen und eine umfassende Empfehlung für die Versorgungsstruktur von spezialisierten IIH-Zentren (AN4IH-Zentren) sowie ein integriertes, interdisziplinäres Diagnose- und Behandlungskonzept für IIH zu geben.
Methodik: Dieser Konsens wurde von einem interdisziplinären Expert*innengremium des AN4IH erstellt, das unter der Schirmherrschaft der Dachgesellschaften für Neurologie (ÖGN), Ophthalmologie (ÖOG), Neuroradiologie (ÖGNR), Neurochirurgie (ÖGNC) und Endokrinologie (ÖGES) eingerichtet wurde und einer formalen Konsensusmethodik folgte.
Resultate: Der AN4IH-Konsensus bietet ein umfassendes Konzept eines integrierten, interdisziplinären Pfades für Versorgungsstruktur, Dringlichkeitseinschätzung, Diagnostik, Therapie und Monitoring sowie Aspekte der Familienplanung und Schwangerschaft bei Patient*innen mit IIH. Der AN4IH-Konsensus ist dabei explizit als Ergänzung bzw. Erweiterung der gültigen Leitlinien zu sehen. Die wesentlichen Inhalte des AN4IH-Konsensus werden im Rahmen eines Kick-off-Meetings während der ÖGN Jahrestagung 2025 präsentiert.
Schlussfolgerung und Ausblick: Mit der Gründung des AN4IH und dem AN4IH-Konsensus wird ein österreichweites interdisziplinäres Netzwerk von spezialisierten Zentren mit einheitlichem Qualitätsstandard geschaffen, das eine flächendeckende State-of-the-Art-Versorgung von Patient*innen mit IIH gewährleistet. Im Rahmen der Erstellung der vorliegenden Konsensus-Empfehlung wurden auch relevante Lücken in der Evidenz in Diagnostik, Therapie und Monitoring der IIH identifiziert, die im Rahmen des AN4IH-Netzwerks mit darin generierten Daten schrittweise geschlossen werden sollen. Ein erster wesentlicher Schritt auf diesem Weg ist die Etablierung einer den Grundsätzen der Datensicherheit entsprechenden und qualitätskontrollierten gemeinsamen Datenbank zur Sammlung sämtlicher klinischer und paraklinischer Daten sowie einer Biobank zur Sammlung von Biosamples.

P97: Gender-related differences in subjects with persisting COVID-19 loss of smell: Baseline data from the SMELL-trial

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Background: Olfactory dysfunction is one of the most common symptoms of COVID-19. Indeed, incidence of loss of smell after SARS-CoV-2 infection has been estimated by meta-analysis to be around 50 %, with subjective recovery

rates ranging from around 3–90 %. Moreover, recent studies have shown that up to 7 % of patients remain anosmic for more than 12 months after the onset of COVID-19 infection.
Research question: Are there gender-related differences in individuals with persisting COVID-19-associated loss of smell?
Method: The SMELL-trial is a monocentric, single blinded, randomized controlled trial evaluating the efficacy of olfactory training in individuals with persisting COVID-19-associated loss of smell (> 3 months post-infection). The individuals with persisting COVID-19-associated loss of smell were recruited at the Department of Neurology at the Medical University of Innsbruck (MUI), Austria. Olfactory performance was measured using the identification and discrimination subscales of the Sniffin' Sticks. Data regarding demographics, comorbidities, quality of life (QoL), mental health, well-being and subjective symptom severity (olfactory visual analogue scale, patient global impression of severity) were collected.
Result: A total of 70 individuals were included. There were more female (64 %) than male (36 %) patients. Mean age was 54 years (SD: ± 14.5) and mean OD duration was 20 months (SD: ± 11.4). No differences between sexes were seen for age, BMI, or symptom duration, neither for comorbidities nor smoking history. There were no gender-related differences for QoL or health, nor for well-being and mood. No differences were seen for subjective symptom severity. However, men compared to women had lower scores on both subscales of the Sniffin' Sticks (identification: 6.85 vs. 9.16, discrimination: 8.81 vs. 10.11, respectively).
Conclusion: In the SMELL-trial cohort of study participants with persisting COVID-19-associated loss of smell, there were no gender-related differences in QoL, well-being, and mood, as well as measures of subjective olfactory impairment, while objective assessment of olfactory function was worse in men compared to women.

P98: Ambulanz für Inklusive Medizin: Ein Modell für barrierefreie Gesundheitsversorgung

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Hintergrund: Die Ambulanz für Inklusive Medizin wurde 2018 ins Leben gerufen, um die gesundheitliche Versorgung von erwachsenen Menschen mit neuronalen Entwicklungsstörungen (z. B. genetische Syndrome wie Trisomie 21, therapierefraktäre früh erworbene Epilepsie, schwere Zerebralparese, erworbene traumatische oder entzündliche Hirnschädigung bei unvollständiger Hirnreifung, frühkindlicher Autismus) zu verbessern. Die ätiologische Grundlage ist vielfältig, mögliche Komorbiditäten über die Lebensspanne sind mannigfaltig und überlappend. Studien zeigen, dass diese Patientengruppe häufig mit strukturellen Barrieren und unzureichendem medizinischem Wissen konfrontiert ist.
Fragestellung: Die zentrale Fragestellung der Ambulanz für Inklusive Medizin lautet: Inwiefern kann eine spezialisierte Einrichtung dazu beitragen, eine bessere Gesundheit und Zufriedenheit dieser Patient*innen zu ermöglichen? Zudem wird die Frage aufgeworfen, wie multidisziplinäre Ansätze in der medizinischen Praxis umgesetzt werden können, um eine optimale Versorgung zu gewährleisten.
Methode: Um diese Fragestellungen zu beantworten, verfolgt die Ambulanz einen interdisziplinären Ansatz. Zum Team mit einer fachärztlichen neurologischen Leitung gehören Pflegekräfte, Psycholog*innen und Sozialarbeiter*innen. Fachärztliche Unterstützung gibt es durch die Psychiatrie. Kooperationspartner im Krankenhaus sind sämtliche andere medizinische Fachrichtungen, insbesondere die Neuroorthopädie und Anästhesie. Diese Fachkräfte arbeiten eng zusammen, um eine ganzheitliche Betreuung zu gewährleisten. Es werden Patienten- und Angehörigenbefragungen und Feedbackgespräche durchgeführt, um die angebotenen Dienstleistungen kontinuierlich zu verbessern.
Ergebnisse: XDie Ergebnisse der Arbeit der Ambulanz für Inklusive Medizin sind vielversprechend. Die Patient*innen und Angehörigen berichten von einer signifikanten Verbesserung ihrer medizinischen Versorgung. Die barrierefreie Gestaltung der Räumlichkeiten und die individuelle Betreuung tragen dazu bei, dass sich die Patient*innen wohl und ernst genommen fühlen. Zudem zeigt sich, dass die interdisziplinäre Zusammenarbeit zu besseren Behandlungsergebnissen führt, da verschiedene Fachrichtungen ihre Expertise einbringen und so eine umfassende Diagnostik und Therapie ermöglichen. Herausforderungen liegen vor allem in der begrenzten Kapazität der Ambulanz und der Notwendigkeit zusätzlicher Ressourcen, um der hohen Nachfrage gerecht zu werden.
Zusammenfassung: Menschen mit neuronalen Entwicklungsstörungen sind eine hochvulnerable Patienten- ▶

gruppe, die aktuell in der medizinischen Versorgung in Österreich oft an ihre Grenzen stößt. Der häufig bestehende erschwerte Zugang zu notwendigen und adäquaten Gesundheitsleistungen gründet sich u. a. auf Fehleinschätzungen im Umgang mit Menschen mit abweichenden oder eingeschränkten Sprach- und Kommunikationsfertigkeiten, auf mangelnden Ausbildungs- und Fortbildungsmöglichkeiten der Ärzteschaft im Studium und in der Facharztausbildung und auf fehlenden strukturellen Rahmenbedingungen. Die Ambulanz für Inklusive Medizin stellt ein innovatives Modell für eine zielgerichtete barrierefreie medizinische Versorgung dar. Unsere Ergebnisse unterstreichen den Bedarf an weiteren spezialisierten Einrichtungen. Perspektivisch könnten ähnliche Konzepte auf regionaler und nationaler Ebene implementiert werden, um eine flächendeckende Verbesserung der Versorgung zu erreichen.

P99: Feasibility and efficacy of a focused neurovascular ultrasound training course for medical students

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Background: Neurovascular ultrasound is an easily applicable bed-side diagnostic tool for guiding the prevention and treatment of cerebrovascular disease. Although sonography training is increasingly integrated in medical school curricula, neurovascular ultrasound has been largely neglected in these efforts.
Research question: We aimed to assess the feasibility and efficacy of a focused neurovascular ultrasound training course for medical students.
Method: Since 2017, small-group neurovascular ultrasound courses were offered for 2nd to 5th-year medical students at the Medical University of Graz, Austria. Demographics and previous ultrasound experience were documented. To test pre-course knowledge, all participants first completed a theoretical test, followed by a 20-hour hands-on training course comprising a standardized step-by-step examination of the extra- and intracranial brain-supplying arteries. Afterwards, all students underwent a practical exam on healthy individuals. The exams were conducted by neurovascular ultrasound experts, blinded to the study's scope and data, using a predefined protocol.
Results: A total of 51 students (median age, 23 years; 24 female, 47 %) participated in the courses. Of those, 27 (53 %) had previous ultrasound experience. Median points of the theoretical pre-course test were 11/30 (IQR: 6). For the practical exam, participants achieved a median score of 56/67 points (IQR: 8). Only 3 students had score levels of < 60 %. Of note, results were independent of the presence of previous ultrasound experience and theoretical pre-course knowledge (p-values each > 0.1).
Summary: This study demonstrates the high efficacy of a simply designed neurovascular ultrasound course for medical students. The findings support the feasibility of integrating neurovascular ultrasound courses into medical school curricula.

P100: Building interprofessional identity in neurology: A randomized controlled trial of interprofessional education

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Background: Interprofessional education (IPE) is an essential aspect of healthcare education, improving patient outcomes through coordinated collaboration. In neurology, where managing complex conditions requires coor-

dinated care among various professionals, IPE is particularly important. Despite its importance, it is rarely included in neurology clerkship curricula or systematically evaluated.

Aim: To assess the impact of a brief, structured IPE intervention on interprofessional identity among medical students.

Objective: To evaluate students' interprofessional identity and to explore their perceptions of interprofessional collaboration and its practical applicability. Additionally, the study aimed to examine the role of IPE in neurology, where interprofessional teamwork is essential.

Method: In this randomized controlled study, 39 neurology clerkship students were allocated either to an interactive 90-minute interprofessional education (IPE) workshop or a non-interactive control session. Data were collected using a post-then-pre design and the German version of the Evaluation of Professional Identity Scale (EPIS-G) questionnaire. The results were analyzed by triangulating EPIS-G scores, perceived challenges and opportunities, and reflections on interprofessional identity and its applicability. Inductive content analyses supported the qualitative evaluation.

Results: 37 students participated in the study, with a mean age of 25 ± 2 years, with 70 % identifying as female. The intervention group (n = 27; mean age 25 ± 1 years) and the control group (n = 10; mean age 27 ± 3 years) had similar gender distributions. The IPE intervention significantly improved interprofessional identity across all EPIS-G domains ($p < 0.001$), while no such changes were observed in the control group. Students identified communication challenges and resource limitations as primary barriers, while recognising opportunities in information sharing and enhanced patient care. Qualitative findings highlighted increased commitment to collaboration, openness to teamwork, and recognition of the patient care benefits of interprofessional practice.

Conclusion: A structured 90-minute IPE workshop within a neurology clerkship can enhance medical students' interprofessional identity. However, sustained and immersive IPE experiences may be required for deeper cognitive shifts, highlighting the complexity of collaborative practice. This study supports the future integration of IPE specifically within neurology to advance collaborative practice.

P101: Neurology data sharing and reuse: Toward the promised land?

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Background: Research funding agencies such as the National Institutes of Health, European Research Agency, and the FWF all promote research data sharing and publishing to the extent possible. Publishers encourage researchers to share their data in order to support their published findings and enhance reproducibility and transparency. Universities are also increasingly promoting research data management and publishing. The rectorate of the University of Graz recently adopted a research data management directive, which requires all researchers to develop data management plans and to publish research data to the maximum extent possible. In this environment, research data repositories (https://www.re3data.org) and data analysis and computational infrastructures such as the European Open Science Cloud are also being developed.

Research question: Our research assesses how neurology-related data is being shared, published, and reused in the context of encouragement and mandates for research data sharing. How is the emerging culture that values data publishing affecting research practices in neurology? How does the published research literature reflect the increasing calls for data publication?

Method: We analyzed the research data repositories from 2 sources: Wikipedia's list of neurology research data repositories and Re3data's neurology repositories, as well as 4 years of papers published in a major European journal (European Journal of Neurology) to scan for mentions of neurology-related data as well as for direct and indirect data citations.

Results: Our findings fall along 2 dimensions: Repositories that self-identify as including neurology-related research data are on the rise. While a similar analysis found 48 repositories on Wikipedia, the list now includes 60 databases. Similarly, Re3data reports an increase in the number of neurology data repositories (118 in 2024 vs. 90 in 2021). In contrast, few published papers directly or indirectly reference published data and almost none cite published data sets.

Conclusion: While the infrastructure for publishing neurology data is advancing with a goal of publishing research data sets for broader availability, the (re)use and citation of such datasets remains low. This mismatch leads us to question the short-term value of published research data and opens up questions regarding the visible and invisible barriers for publishing and reuse of research data in neurology.

P102: Signs of intracranial hypertension in chronic inflammatory polyradiculoneuropathies: A cross sectional cohort study

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Background: Inflammatory polyradiculoneuropathy (IPN) has been associated with intracranial hypertension (IH) in various case reports, suggesting a potential link between the two conditions.
Objective: The aim of this prospective study was to investigate the prevalence of typical signs of IH in a population of patients with IPN.
Method: In this cross-sectional prospective study, we prospectively screened consecutive patients with chronic IPN for the presence of clinical and paraclinical signs of IH (fundoscopy, perimetry, optical coherence tomography, ultrasonography) between August 31 2021 and December 31 2023.
Results: Of 27 patients included (median age: 61.0 years [IQR 22.0]; 30.8 % female; median time since diagnosis: 49.5 months [IQR 42.0]), 10 patients (37.0 %) with IPN had sonographic evidence of IH (IPN+IH). Compared to patients without signs of IH, IPN+IH were younger (40.5 years [24.0] vs. 63.0 years [10.0]) and had higher CSF total protein (67.7mg/dl [57.6] vs. 56.1mg/dl [26.7]). However, IPN+IH did not have higher BMI (24.1 [5.2] vs. 26.0 [3.8]) or female predominance (33.3 % vs. 29.4 %).
Discussion: Signs of IH seem a common feature in IPN, indicating a likely association. The demographic and clinical profile of IPN+IH patients differs from that of idiopathic IH, suggesting that IH in IPN is likely secondary and may be linked to elevated CSF protein. Given that most patients were asymptomatic and that the specificity of IH symptoms remains low, routine screening for subclinical IH signs may be warranted in this population.

P103: Cognitive performance in individuals at risk for Parkinson's disease: Preliminary results from the prospective population-based HeBA Tyrol Study

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Background: Research indicates that cognitive functions may be impaired years before the clinical diagnosis of Parkinson's disease (PD). The Healthy Brain Aging (HeBA) project is a longitudinal, multicenter initiative aimed at investigating risk factors, including cognitive changes, associated with the development of PD.
Objective: To investigate potential cognitive changes in a population-based cohort at high risk for PD.
Method: The HeBA project implemented an online questionnaire designed to identify risk factors and prodromal indicators for PD. This included high-interest single questions (HIQs) related to smell loss, dream enactment behavior, family history of PD, and subjective cognitive decline, in addition to validated assessment scales. A subset of participants underwent a remote olfactory assessment using the University of Pennsylvania Smell Identification Test (UPSIT), which was followed by detailed in-person assessments for another subset of participants. The in-person visits included, among other assessments, neuropsychological tests (Montreal Cognitive Assessment [MoCA], Consortium to Establish a Registry for Alzheimer's Disease [CERAD-plus], symbol digit modalities test [SDMT]), and questionnaires on non-motor symptoms (NMS). Here, we present a preliminary analysis of the HeBA Tyrol sample, with in-person assessments beginning in March 2023. Participants were classified as high- or low-risk for PD based on information from the initial online survey and results from the UPSIT. High-risk individuals

scored below the 10th percentile of age- and sex-scaled norms on the UPSIT and were positive for family history of PD and REM sleep behavior disorder symptoms.

Results: Of the 116 subjects assessed between March 2023 and February 2024 (mean age 63 ± 5 years, 67 % female), 65 participants (56 %) were classified as high-risk, 51 (44 %) as low-risk. The high-risk group reported a higher burden of NMS (Mann-Whitney tests, Ps < .05). Subjective memory complaints were more frequent in the high-risk group (22 % vs. 4 %; Chi-square test, $p < 0.006$). High-risk participants showed lower objective performance than low-risk participants in verbal learning only (Mann-Whitney test, $p = 0.029$). In a regression analysis, subjective memory complaints and verbal learning were significant predictors of group classification between high- and low-risk groups (Nagelskerke $R^2 = 0.14$, $p = 0.001$). By adding ratings of depressive symptoms to the model, verbal learning remained the only significant predictor (Nagelskerke $\Delta\text{-}R^2 = 0.03$, $p = 0.109$).

Conclusion: Compared with low-risk participants, high-risk individuals exhibit significant changes in both subjective and objective cognitive measures. Longitudinal evaluations in the larger multicenter HeBA project will further elucidate cognitive changes in prodromal PD. Updated analyses from a larger sample of participants assessed through December 2024 will be presented at the congress.

P104: Einnahme fester Medikamente bei schlaganfallassoziierter Dysphagie: Erkenntnisse aus Fragebogenerhebungen von Pflegekräften und Logopäd*innen

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Hintergrund: Die Entwicklung einer Dysphagie zählt zu den häufigsten Komplikationen nach einem Schlaganfall und betrifft bis zu 75 % der Patient*innen in der Akutphase. Neben der Entscheidung über die adäquate Nahrungsaufnahme stellt besonders die sichere Verabreichung fester Medikamente eine interdisziplinäre Herausforderung dar. Trotz der Relevanz fehlen standardisierte evidenzbasierte Leitlinien, was zu Unsicherheiten und uneinheitlichen Ansätzen in der Versorgung führt.

Fragestellung: Wie bewerten Pflegekräfte und Logopäd*innen die Praxis der Medikamenteneinnahme bei Patient*innen mit schlaganfallassoziierter Dysphagie? Welche Methoden werden verwendet, und welche Herausforderungen werden dabei identifiziert? Ziel dieser Umfragen war es, die Handhabung und Herausforderungen bei der Einnahme fester Medikamente von Schlaganfallpatient*innen mit Dysphagie in der klinischen Praxis zu beschreiben.

Methode: Im Rahmen zweier Online-Umfragen wurden a) Pflegekräfte (n = 195) und b) Logopäd*innen (n = 147) in deutschsprachigen Ländern zu Screening-Methoden, Entscheidungsprozessen und Handlungsstrategien bei der Medikamentengabe befragt. Die Daten wurden berufsgruppenspezifisch analysiert, wobei zusätzlich gemeinsame Herausforderungen und Schnittstellen identifizieren wurden.

Ergebnisse: – Pflegekräfte: 50,8 % der Befragten auf Stroke Units nutzten standardisierte Dysphagie-Screenings, jedoch gaben 92,6 % an, feste Medikamente bei Verdacht auf Dysphagie zu modifizieren, zumeist durch Zerkleinern. Begleitboli wie Apfelmus und verdicktes Wasser wurden häufig verwendet, wobei Unsicherheiten hinsichtlich ihrer Eignung und Sicherheit berichtet wurden.

– Logopäd:innen: 73,5 % bewerteten die Schluckfähigkeit fester Medikamente im klinischen Alltag, meist durch die Nutzung patienteneigener Medikamente. Entscheidungsgrundlagen für die Modifikation basierten auf klinischen Symptomen wie Husten oder oralen Resten, jedoch fehlten häufig standardisierte Bewertungsmaßstäbe.

Zusammenfassung: Die Ergebnisse zeigen, dass Unsicherheiten und methodische Inkonsistenzen sowohl bei Pflegekräften als auch Logopäd*innen bestehen, insbesondere bei der Modifikation fester Medikamente und der Auswahl geeigneter Begleitboli. Trotz der Anwendung von Screenings bleibt die Entscheidungsfindung oft subjektiv und uneinheitlich. Es besteht ein dringender Bedarf an interdisziplinären Fortbildungsprogrammen und evidenzbasierten Leitlinien, um die Sicherheit der Medikamenteneinnahme bei schlaganfallassoziierter Dysphagie zu verbessern und die klinische Praxis zu vereinheitlichen.

P105: Immuncheckpoint-Inhibitor-assoziierte ausgedehnte longitudinale transverse Myelitis

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Anamnese: Eine 50-jährige Patientin mit einem 2020 diagnostizierten malignen Melanom (Stadium IIIB, pT2b, N0, M0) im Bereich des rechten Oberschenkels erhielt nach der initialen kompletten Resektion 8 Zyklen einer Immuncheckpoint-Inhibitor-(ICI-)Therapie mit Pembrolizumab (Anti-Programmed-Cell- Death-(PD)-1-IgG4-Antikörper).

Klinische Präsentation: Die initiale klinische Präsentation im Mai 2024 erfolgte in Form von über 4 Wochen aufsteigenden Gefühlsstörungen und subjektivem Kältegefühl beider Beine, zuletzt mit Einbeziehung des Rumpfes bis unter die Mamille bds reichend. Unter der Annahme einer ICI-assoziierten Polyneuropathie wurde eine Therapie mit Pregabalin und Prednisolon etabliert, womit eine moderate Verbesserung der Symptome erzielt werden konnte. 1 Monat später kam es aber zu einer Verschlechterung des Gangbildes und Harninkontinenz bei gleichbleibender Ausprägung der vorbestehenden sensiblen Defizite. Zu diesem Zeitpunkt hatte die Patientin eine links-betonte Paraparese KG 3, eine Ataxie, vermindertes Vibrationsempfinden und Oberflächensensibilität bd Beine.

Abklärung und interdisziplinäre Falllösung: Initial wurden eine Lumbalpunktion (LP) sowie ein MRT des Schädels und der gesamten Wirbelsäule durchgeführt.

In der LP wurden eine lymphozytäre Pleozytose mit 39 Zellen/µl und ein erhöhter Gesamteiweißgehalt von 82 mg/dl diagnostiziert. In der wiederholten LP 1 Monat später fand man eine lymphomonozytäre Pleozytose mit 246 Zellen/µl, darunter vereinzelt Plasmazellen, bei einem unveränderten Gesamteiweißgehalt des Liquors und unveränderter leichter Blut-Hirn-Schrankenstörung. Tumorzellen konnten in beiden Proben nicht nachgewiesen werden. Multiple und wiederholte Testungen konnten keinen Erreger nachweisen. Auch alle onkoneuronalen und Oberflächen-Antikörper im Serum und Liquor waren negativ. CXCL13-Levels waren erhöht.

Die MRT der Wirbelsäule zeigte eine T2/FLAIR-Signalhyperintensität beinahe der gesamten Zirkumferenz des Myelons mit Kontrastmittelaufnahme von C3 bis Th5 reichend. Die Verdachtsdiagnose einer longitudinalen transversen Myelitis (LETM) als Nebenwirkung der ICI-Therapie wurde geäußert. Hochdosis-Methylprednisolon über 5 Tage brachte eine leichte Besserung der Paresen (KG 3-4) und der Gefühlsstörungen bei gleichbleibender Stuhl- und Harninkontinenz.

Während der 1-wöchigen Anschluss-Rehabilitationsphase kam es zu einer Verschlechterung der Paraparese mit Schmerzzunahme zervikothorakal. Diesmal hatte die Patientin eine Paraplegie, ein sensibles Level Th7, fehlende Patellarsehnenreflexe bds bei kloniform gesteigertem Achillessehnenreflex rechts und Dysphagie. Diesmal zeigte die MRT der Wirbelsäule inkl. kraniozervikalem Übergang ein diffuses Ödem des gesamten Rückenmarks inkl. Medulla oblongata mit zentral betonter Kontrastmittel-Aufnahme, vereinbar mit der Diagnose LETM. Die Hochdosis Methylprednisolon-Pulstherapie wurde eskaliert und insgesamt 7 Tage verabreicht. Aufgrund fehlender Wirksamkeit wurden 7 Zyklen Plasmapherese angeschlossen. Die Paraparese und das sensible Niveau besserten sich (KG max. 3/5). Orales Methylprednisolon 75 mg wurde bei Entlassung langsam ausschleichend in der Dosis reduziert.

Take-Home Message: ICI-assoziierte LETM ist eine seltene, doch gravierende Nebenwirkung einer hochwirksamen Therapie des malignen Melanoms. Standardbehandlungspfade gibt es bisher nicht. Unter intravenöser Methylprednisolon-Pulstherapie kann es zu einer vorübergehenden Besserung der Symptome, gefolgt von einer gravierenden Verschlechterung, kommen. In solchen Fällen können die Eskalation der Methylprednisolon-Dosis und die Behandlungsdauer sowie die Plasmapherese zu einer deutlichen Verbesserung der neurologischen Defizite führen.

P106: Integrating physiotherapy and disease-modifying therapy in spinal muscular atrophy type III: A case report of multidisciplinary rehabilitation

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Medical history: A 62-year-old female with spinal muscular atrophy (SMA) type III and severe proximally accentuated flaccid tetraparesis, wheelchair-dependent since childhood, commenced disease-modifying therapy (DMT) with risdiplam at the standard dose in December 2021. Under DMT, her motor performance stabilised. Physiotherapy was initiated in June 2023 to address persistent functional deficits. Comorbidities included arterial hypertension and hypothyroidism.

Clinical presentation: The patient presented with significant postural instability, asymmetric upper limb weakness, and limited endurance. These impairments negatively impacted her daily activities, including knitting and computer use.

Diagnostic process: A comprehensive neurophysiotherapeutic and neurological evaluation identified severe motor and postural dysfunction. Functional impairments were quantified using the Revised Upper Limb Module (RULM), with scores of 16 (right) and 14 (left), and a CHOP ANTEND (Children's Hospital of Philadelphia Adult Test of Neuromuscular Disorders) score of 28 at start of physical therapy. Muscle strength testing revealed distal muscle function graded as Medical Research Council (MRC) 3 in the wrist and ankle muscles, with a progressive proximal decline reaching MRC 1 in the shoulder and hip girdles. Radiological imaging demonstrated an S-shaped scoliotic curvature of the thoracic spine, severe left-convex rotational lumbar scoliosis with degenerative changes, and right hip dysplasia. Additionally, inadequate support in the patient's wheelchair was identified as a significant factor exacerbating postural instability and functional limitations.

Interdisciplinary management: The patient participated in a 12-week physiotherapist-led programme at the Department of Neurology of the Medical University of Vienna, targeting motor and postural impairments. Visual stabilization exercises using a laser pointer were implemented to improve head control, while proprioceptive neuromuscular facilitation (PNF) techniques addressed motor coordination in the pelvis, shoulders, and head. Resistance bands were utilised to improve head control, and pull machines were employed to strengthen both the upper and lower limbs. Endurance training for the extremities was performed at home 4 times weekly on a MOTOmed ergometer in a wheelchair. Training intensity was monitored using the visual version of the modified Borg scale (0–10). Disease progression and clinical response to risdiplam were monitored. A customised seating system, adapted in collaboration with a certified technician, improved head and trunk stability, reducing the patient's effort to maintain posture during therapy and daily activities. Adherence to the rehabilitation plan was supported by a caregiver who assisted with transport and home-based exercises.

Following the 12-week programme, the patient reported improved head stability during wheelchair use and successfully achieved personal goals, including increased endurence during knitting and computer use, as measured with an activity log. RULM scores changed from 16 (right) and 14 (left) to 16 bilaterally. A change of 2 points is considered clinically meaningful. CHOP ATEND scores improved from 28 to 34.

Take-home message: XThis case demonstrates the feasibility and potential efficacy of a multidisciplinary approach to managing SMA type III. A structured neurological care plan, tailored physiotherapy, personalised wheelchair adjustments, and caregiver involvement collectively improved motor function, postural stability, and daily activities. This case underscores the importance of integrated, patient-centerd strategies in the rehabilitation of adults with SMA.

P107: Kopfschmerzen und Wesensveränderung bei unklarer Meningoenzephalitis – Kein Sommer wie damals für zwei Patienten im August 2024

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Anamnese: Bei Fall 1 handelt es sich um einen 51-jährigen Patienten mit einem 4 Monate zuvor diagnostizierten Hodgkin-Lymphom, unter abgeschlossener Chemotherapie in Remission. Ab August 2024 kam es bei dem Patienten zu rezidivierenden Episoden mit Kopfschmerzen, Erbrechen und Abdominalgien, Fieberschüben mit unklarem Fokus sowie qualitativen und quantitativen Bewusstseinsänderungen. Im weiteren Verlauf klagte er über Schmerzen und Krämpfe der Muskulatur bei Anstrengung im Bereich der Arme und des Beckengürtels.
Fall 2 beschreibt einen 66-jährigen Patienten, der ebenso mit Kopfschmerzen und Wesensveränderung im August 2024 aufgenommen wurde. Zudem klagte er über vermehrte Schläfrigkeit und Gangunsicherheit. Vorerkrankungen waren eine arterielle Hypertonie und ein Vorhofflimmern.
Klinische Präsentation: Patient 1 zeigte eine stark fluktuierende Bewusstseinstrübung (von wach-apathisch bis soporös) mit Fieber und Meningismus. Im weiteren Verlauf waren progrediente Paresen und Atrophien der kleinen Handmuskeln beidseits fassbar.
Patient 2 wies eine psychomotorische Verlangsamung und subjektive Gangunsicherheit auf, wobei im neurologischen Status keine motorischen oder sensiblen Ausfälle noch Ataxie sichtbar waren.
Abklärung: In beiden Fällen zeigte die zerebrale Bildgebung mittels MRT inklusive Kontrastmittelgabe keine wegweisenden Befunde. Bei beiden Patienten fand sich in der Liquordiagnostik eine Pleozytose von 61 bzw. 81 Zellen/µl mit lymphomonozytärem Zellbild und erhöhtem Gesamteiweiß, ohne intrathekale Immunglobulin-Synthese. Eine umfassende Diagnostik für häufige neurotrope Erreger (Breitspektrum-PCR BioFire®, Liquorkultur, Borrelien-Serologie im Liquor) sowie die antineuronalen Antikörper zum Ausschluss einer autoimmunvermittelten Enzephalitis waren jeweils negativ. In beiden Fällen auffällig waren wiederholte EEGs mit Zeichen einer generalisierten Verlangsamung und frontal intermittierender rhythmischer Delta-Aktivität.
Interdisziplinäre Falllösung: Als weitere mögliche Manifestation zeigte Patient 1 auffällige elektrophysiologische Befunde mit neurografisch prädominanter Reduktion der motorischen Amplituden, mehr an den oberen als an den unteren Extremitäten, bei elektromyografisch subakut neurogenem Schädigungsmuster.
Patient 2 wiederum klagte im Verlauf der Erkrankung über verschwommenes Sehen, was ophthalmologisch auf eine Panuveitis zurückzuführen war und mit lokaler und systemischer oraler Cortisontherapie gut behandelt werden konnte.
Take-Home Message: Insbesondere in den Sommermonaten bis Frühherbst soll beim klinischen Bild einer Meningoenzephalitis und unklarer Liquorpleozytose an die Möglichkeit dieser Erkrankung gedacht und eine entsprechende Diagnostik veranlasst werden. Darüber hinaus soll beachtet werden, dass über eine Meningoenzephalitis hinaus noch andere Manifestationen möglich sind, wie unter anderem eine Poliomyelitis-ähnliche Symptomatik oder Augenbeteiligung. Relevant ist auch die epidemiologische Entwicklung dieser Erkrankung, da in Österreich seit 2010 nur Einzelfälle erfasst wurden, im Jahr 2024 allerdings so viele Fälle wie noch nie zuvor verzeichnet wurden und wahrscheinlich ein weiterer Anstieg bzw. zumindest ein ähnlich hohes Niveau in den nächsten Jahren zu erwarten ist.

P108: Postiktale MRT-Veränderungen – Eine seltene Differenzialdiagnose zur Encephalitis

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Anamnese: Ein 54-jähriger Patient wurde verwirrt von Passanten aufgefunden und per Polizei und Notarzt an die Notaufnahme des Universitätsklinikums Tulln gebracht. Eine genaue Fremdanamnese war nicht erhebbar, eine Eigenanamnese nicht möglich; es wird lediglich berichtet, dass der Patient durch Ratlosigkeit auffiel, als er versuchte, Geld zu beheben.
Klinische Präsentation: In der

initialen Beurteilung präsentierte sich der Patient stark agitiert, verbal nicht erreichbar und mit stereotypen Antworten. Nach pharmakologischer Therapie der Hyperaktivität demaskierte sich eine ausprägte Störung des Sprachverständnisses, eine Aufmerksamkeitsstörung und eine kognitive Störung mit retrograder Amnesie für mehrere Tage sowie für andere Gedächtnisinhalte, u. a. den Handy-PIN.

Abklärung: Im Akut-EEG zeigte sich anhaltend rhythmische Aktivität über links temporal im Sinne eines fokalen Status epilepticus. Nach Anfallsunterbrechung erfolgte eine zerebrale MRT, die multifokale hyperintense Läsionen in den DWI und T2-Sequenzen unter anderem links kortikal und subkortikal sowie parahippocampal links zeigte. Diese wurden initial als ischämisch, differenzialdiagnostisch im Rahmen einer Encephalitis interpretiert. In der Lumbalpunktion fand sich eine unauffällige Zellzahl von 3 Zellen (0–5 Zellen/µl) und erhöhtes Gesamtprotein von 1.801 mg/l (180–430 mg/l), bei sonst unauffälliger Erreger-Diagnostik. Ein St. p. Lues-Infektion war bereits aus den Vordiagnosen bekannt und wurde bestätigt.

In Verlaufs-EEG-Kontrollen kam es bis auf intermittierende Verlangsamungswellen links temporal zur Normalisierung des EEG-Befundes. Im zerebralen MRT am Tag 4 wurde ein Rückgang der MR-Veränderungen ohne neu auftretende Läsionen festgestellt. Innerhalb von gesamt 10 Tagen kam es zur kompletten Remission der neurologischen und neuropsychologischen Zeichen.

Interdisziplinäre Falllösung: Anhand der Schädel-MRT-Befunde wurde die Verdachtsdiagnose eines zytotoxischen Ödems im Rahmen eines Status epilepticus gestellt.

MRT-Veränderungen bei Patienten mit Status epilepticus sind ein seltenes Phänomen, das aufgrund der Ähnlichkeit zu möglichen Auslösern eines Status epilepticus, z. B. Ischämie, Encephalitis oder Tumorerkrankung, eine weitere Abklärung erfordert. Auch seltenere Differenzialdiagnosen wie CHANTER-Syndrom nach opiodbedingter Neurotoxizität oder PRES bei Hypertension sind zu bedenken.

In der Literatur sind unter anderem DWI- und T2-hyperintense Läsionen, T1-Hypointensität sowie Kontrastmittelaufnahmen beschrieben. Typische Lokalisationen umfassen kortikale, subkortikale und hippocampale Läsionen sowie Klaustrum und Splenium. Eine Assoziation zur betroffenen Seite im EEG ist ebenfalls beschrieben.

Take-Home-Message: Zytotoxische Ödeme durch epileptische Anfälle sowie im Status epilepticus sind ein wichtiges, aber seltenes radiologisches Zeichen in der klinischen Praxis und abzugrenzen von Ischämien und Encephalitiden bzw. auch PRES oder CHANTER Syndrom. Nach adäquater antikonvulsiver Behandlung kommt es in der Regel zu einer (Teil-)Remission der Symptome und cMRT-Zeichen.

P109: Gekreuzte Hirnnervenparesen und schlaffe Paraparese der Arme

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Anamnese: Eine 82-jährige Patientin wird vorstellig mit einer Schwäche der rechten oberen Extremität und einer Ptose links, beides seit 1 Woche bestehend.

An relevanten Vorerkrankungen besteht ein diffuses großzelliges B-Zell-Lymphom mit 6 stattgehabten Zyklen Immun(chemo)therapie nach R-CHOP-Schema, letzte Gabe vor 2 Monaten.

Klinische Präsentation: Bei Aufnahme zeigt sich eine reinmotorische schlaffe Parese Kraftgrad 3 der rechten oberen Extremität, eine komplette Ptose links mit eingemauertem Bulbus, Mydriasis und fehlender Lichtreaktion. Im Verlauf entwickelt die Patientin zudem rasch eine periphere Fazialisparese rechts mit Lidschlussdefizit von 2 mm sowie eine reinmotorische schlaffe Parese der linken oberen Extremität.

Abklärung: Die zerebrale MRT schließt Ischämie, Blutung oder Raumforderung aus, die HWS-MRT zeigt multisegmentale Diskopathien mit beidseitigen Neuroforamenstenosen C4–C7. Die Lumbalpunktion ergibt einen gänzlich unauffälligen Befund mit negativer Erregerdiagnostik.

Elektrophysiologisch finden sich Leitungsblöcke über dem Erb-Punkt beidseits bei insgesamt axonaler Läsion der Armnerven, zudem fehlende F-Wellen des N. medianus beidseits und N. ulnaris rechts sowie schlecht ausgeprägte F-Wellen über dem N. ulnaris links.

Bei Verdacht auf multifokale motorische Neuropathie erhält die Patientin eine 5-tägige Immunglobulintherapie, jedoch ohne klinische Besserung; nebenbefundlich tritt eine Leukopenie auf.

Weiters erfolgt eine Thorax-CT, die keine Raumforderung abbildet. ▶

Interdisziplinäre Falllösung: Rückblickend war die Patientin bereits 1 Jahr davor mit einer Gangstörung, vertikaler Blickparese nach oben, verlangsamten Sakkaden nach unten und mildem linksbetontem Parkinson-Syndrom vorstellig. Bei Verdacht auf progressive supranukleäre Blickparese wurde das klinisch gut wirksame PK-Merz etabliert, das im Laufe von Folgekrankenhausaufenthalten verloren ging. Madopar zeigte keine Wirksamkeit. Ein DAT-Scan zeigte keinen Nachweis einer Degeneration der nigrostriatalen Bahnen.

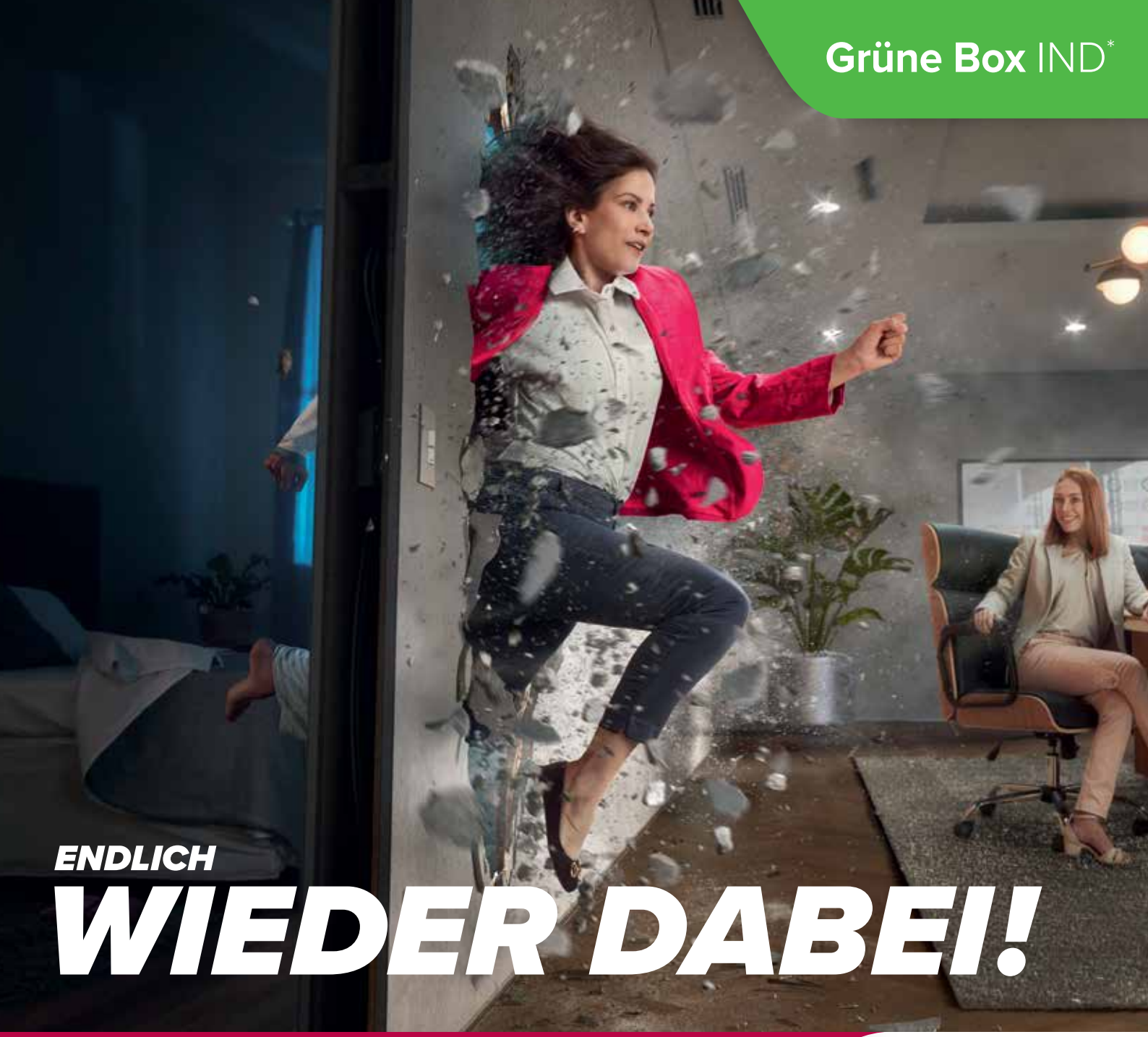
Wenige Monate danach wurde schließlich aufgrund einer milden Panzytopenie in einem auswärtigen Krankenhaus ein B-Zell-Lymphom diagnostiziert, das auf die Therapie nach R-CHOP Schema laut betreuender Onkologie gut ansprach.

3 weitere Monate später wurde die Patientin hierorts akut mit Doppelbildern und einer äußeren Oculomotoriusparese links aufgenommen. Mehrfache cMRTs zeigten keine Ischämie, die Lumbalpunktion war unauffällig ohne Nachweis onkoneuronaler, Gangliosid- oder neuronaler Rezeptor-Oberflächenantikörper.

Klinisch kommt es im rezenten Verlauf zu einer progredienten Symptomatik mit gekreuzten Hirnnervenparesen und schlaffer reinmotorischer Paraparese beider Arme, sodass eine PET-CT urgiert wird. Dabei zeigen sich multiple FDG-avide Lymphknoten beidseits des Zwerchfells und im rechten Mastoid, zudem Orbitabeteiligung links. Der Befund entspricht einem Progress der Tumorerkrankung. Rückblickend zeigte bereits ein auswärtiges PET-CT ein halbes Jahr zuvor multiple FDG-avide Läsionen, darunter in der linken Orbita nahe dem M. rectus lat., im Adenoid rechts sowie zahlreiche zervikale, nuchale, supra- und infradiaphragmale Lymphknoten. Zusätzlich waren beidseitige Nierenbefunde sowie auffällige Glandulae parotis, submentale und sublinguale Drüsen zu sehen.

Damit sind die periphere Fazialisparese rechts, der eingemauerte Bulbus links mit Ptose und die schlaffe Paraparese der Arme durch lokale infiltrative Prozesse zu erklären.

Take-Home Message: Bei der Patientin zeigt sich eine komplexe progrediente neurologische Symptomatik, die initial differenzialdiagnostisch sowohl entzündlich, ischämisch als auch neurodegenerativ abgeklärt wurde. Nach Ausschluss anderer Ursachen und fehlendem Ansprechen auf eine Immunglobulintherapie ergab ein PET-CT multiple FDG-avide Läsionen, die auf einen Progress des bekannten B-Zell-Lymphoms hinweisen. Die aktuellen klinischen Befunde, einschließlich gekreuzter Hirnnervenparesen und schlaffer Paraparese der oberen Extremitäten, deuten auf eine infiltrative Genese hin, die eine onkologische Behandlung erfordert.



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EPTI-0136, 12/2024 Fachkurzinformation siehe Seite XXX